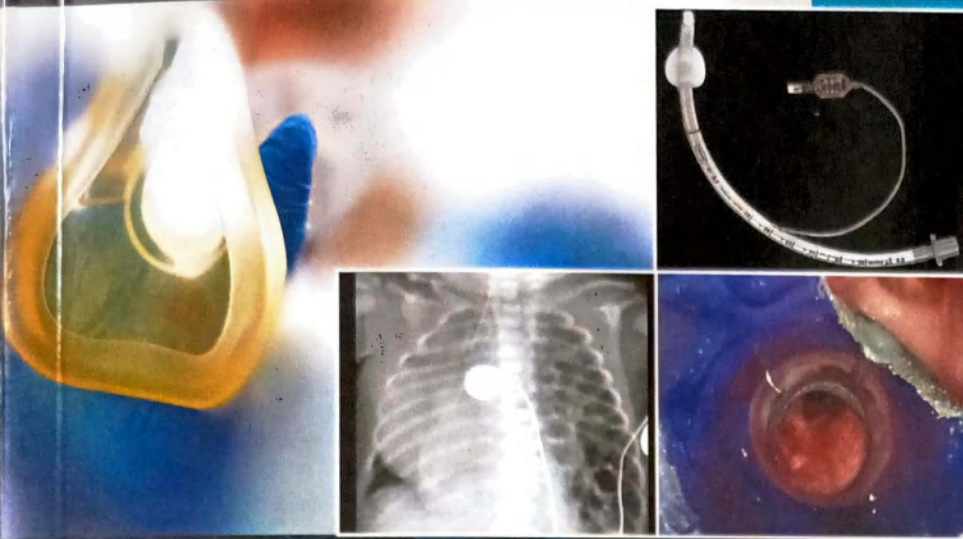




5th Edition

Objective ANESTHESIA Review

A Comprehensive Textbook for the Examinees

*Under the aegis of
Department of Anesthesiology
Critical Care & Pain
Tata Memorial Hospital*



**Atul Prabhakar Kulkarni
Jigeeshu Vasistha Divatia
Vijaya Prakash Patil**

Foreword
RA Badwe



**Objective
ANESTHESIA
Review**

A Comprehensive Textbook for the Examinees

Objective ANESTHESIA Review

A Comprehensive Textbook for the Examinees

Fifth Edition

Editors

Atul Prabhakar Kulkarni

MD (Anesthesiology) FISCCM PGDHHM FICCM
Professor and Head
Division of Critical Care
Department of Anesthesiology
Critical Care and Pain
Tata Memorial Hospital
Homi Bhabha National Institute
Mumbai, Maharashtra, India

Jigeeshu Vasistha Divatia

MD (Anesthesiology) FICCM FCCM
Professor and Head
Department of Anesthesiology
Critical Care and Pain
Tata Memorial Hospital
Homi Bhabha National Institute
Mumbai, Maharashtra, India

Vijaya Prakash Patil

MD (Anesthesiology) Dip (Hosp Adminis)
Professor
Department of Anesthesiology
Critical Care and Pain
Tata Memorial Hospital
Homi Bhabha National Institute
Mumbai, Maharashtra, India

Foreword

RA Badwe



JAYPEE BROTHERS MEDICAL PUBLISHERS

The Health Sciences Publisher

New Delhi | London



Jaypee Brothers Medical Publishers (P) Ltd

Headquarters

Jaypee Brothers Medical Publishers (P) Ltd
4838/24, Ansari Road, Daryaganj
New Delhi 110 002, India
Phone: +91-11-43574357
Fax: +91-11-43574314
E-mail: jaypee@jaypeebrothers.com

Overseas Offices

JP Medical Ltd
83 Victoria Street, London
SW1H 0HW (UK)
Phone: +44 20 3170 8910
Fax: +44 (0)20 3008 6180
E-mail: info@jpmedpub.com

Website: www.jaypeebrothers.com

Website: www.jaypeedigital.com

© 2021, Jaypee Brothers Medical Publishers

The views and opinions expressed in this book are solely those of the original contributor(s)/author(s) and do not necessarily represent those of editor(s) of the book.

All rights reserved. No part of this publication may be reproduced, stored or transmitted in any form or by any means, electronic, mechanical, photocopying, recording or otherwise, without the prior permission in writing of the publishers.

All brand names and product names used in this book are trade names, service marks, trademarks or registered trademarks of their respective owners. The publisher is not associated with any product or vendor mentioned in this book.

Medical knowledge and practice change constantly. This book is designed to provide accurate, authoritative information about the subject matter in question. However, readers are advised to check the most current information available on procedures included and check information from the manufacturer of each product to be administered, to verify the recommended dose, formula, method and duration of administration, adverse effects and contraindications. It is the responsibility of the practitioner to take all appropriate safety precautions. Neither the publisher nor the author(s)/editor(s) assume any liability for any injury and/or damage to persons or property arising from or related to use of material in this book.

This book is sold on the understanding that the publisher is not engaged in providing professional medical services. If such advice or services are required, the services of a competent medical professional should be sought.

Every effort has been made where necessary to contact holders of copyright to obtain permission to reproduce copyright material. If any have been inadvertently overlooked, the publisher will be pleased to make the necessary arrangements at the first opportunity. The **CD/DVD-ROM** (if any) provided in the sealed envelope with this book is complimentary and free of cost.

Not meant for sale.

Inquiries for bulk sales may be solicited at: jaypee@jaypeebrothers.com

Objective Anesthesia Review: A Comprehensive Textbook for the Examinees

First Edition: 2009

Second Edition: 2012

Third Edition: 2013

Fourth Edition: 2017

Fifth Edition: 2021, Reprint: 2023

ISBN 978-93-900-20-48-5

Printed at: Samrat Offset Pvt. Ltd.

Dedicated to

The examinees, may this book solve (most of) your examination problems!

Contributors

Amol Trimbakrao Kothekar MD (Anesthesiology)
Associate Professor
Department of Anesthesiology, Critical Care and Pain
Tata Memorial Hospital
Mumbai, Maharashtra, India

Aparna Chatterjee MD (Anesthesiology) FRCA
Professor
Department of Anesthesiology, Critical Care and Pain
Tata Memorial Hospital
Mumbai, Maharashtra, India

Atul Prabhakar Kulkarni
MD (Anesthesiology) FISCCM PGDHHM FICCM
Professor and Head
Division of Critical Care
Department of Anesthesiology
Critical Care and Pain
Tata Memorial Hospital
Homi Bhabha National Institute
Mumbai, Maharashtra, India

Bhakti Trivedi MD (Anesthesiology)
Associate Professor
Department of Anesthesiology, Critical Care and Pain
Tata Memorial Hospital
Mumbai, Maharashtra, India

Bindiya Salunke MD (Anesthesiology)
Assistant Professor
Department of Anesthesiology, Critical Care and Pain
Tata Memorial Hospital
Mumbai, Maharashtra, India

Debashree Lahiri DNB (Anesthesiology)
Assistant Professor
Department of Anesthesiology
Critical Care and Pain
Tata Memorial Hospital
Mumbai, Maharashtra, India

Jeson Rajan Doctor MD (Anesthesiology)
Associate Professor
Department of Anesthesiology
Critical Care and Pain
Tata Memorial Hospital
Mumbai, Maharashtra, India

Jigeeshu Vasistha Divatia MD (Anesthesiology) FICCM FCCM
Professor and Head
Department of Anesthesiology
Critical Care and Pain
Tata Memorial Hospital
Homi Bhabha National Institute
Mumbai, Maharashtra, India

Madhavi Desai MD (Anesthesiology)
Associate Professor
Department of Anesthesiology, Critical Care and Pain
Tata Memorial Hospital
Mumbai, Maharashtra, India

Madhavi Shetmahajan MD (Anesthesiology) FRCA
Professor
Department of Anesthesiology, Critical Care and Pain
Tata Memorial Hospital
Mumbai, Maharashtra, India

Malini Joshi MD (Anesthesiology)
Professor
Department of Anesthesiology, Critical Care and Pain
Tata Memorial Hospital
Mumbai, Maharashtra, India

Nayana Amin MD (Anesthesiology)
Professor
Department of Anesthesiology, Critical Care and Pain
Tata Memorial Hospital
Mumbai, Maharashtra, India

Parmanand Jain MD (Anesthesiology)
Professor
Department of Anesthesiology, Critical Care and Pain
Tata Memorial Hospital
Mumbai, Maharashtra, India

Priya Ranganathan MD (Anesthesiology)
Professor
Department of Anesthesiology, Critical Care and Pain
Tata Memorial Hospital
Mumbai, Maharashtra, India

Raghu S Thota MD (Anesthesiology) MNAMS
Fellowship in Pain Management (Singapore)
Associate Professor
Department of Anesthesiology, Critical Care and Pain
Tata Memorial Hospital
Mumbai, Maharashtra, India

Reshma Ambulkar MD (Anesthesiology) FRCA
Professor
Department of Anesthesiology, Critical Care and Pain
Tata Memorial Hospital
Mumbai, Maharashtra, India

Sheila Nainan Myatra MD (Anesthesiology)
Professor
Department of Anesthesiology
Critical Care and Pain
Tata Memorial Hospital
Mumbai, Maharashtra, India

Shilpushp Jagannath Bhosale MD (Anesthesiology)
Assistant Professor
Department of Anesthesiology, Critical Care and Pain
Tata Memorial Hospital
Mumbai, Maharashtra, India

Sohan Lal Solanki MD PDCC MAMS
Associate Professor
Department of Anesthesiology, Critical Care and Pain
Tata Memorial Hospital
Mumbai, Maharashtra, India

Sudivya Sharma MD DNB
Fellowship in Pediatric Anesthesia
Assistant Professor
Department of Anesthesiology, Critical Care and Pain
Tata Memorial Hospital
Mumbai, Maharashtra, India

Sukhada Savarkar MD
Assistant Professor
Department of Anesthesiology, Critical Care and Pain
Tata Memorial Hospital
Mumbai, Maharashtra, India

Sumitra Ganesh Bakshi MD (Anesthesiology)
Professor
Department of Anesthesiology, Critical Care and Pain
Tata Memorial Hospital
Mumbai, Maharashtra, India

Swapnil Parab MD (Anesthesiology)
Associate Professor
Department of Anesthesiology, Critical Care and Pain
Tata Memorial Hospital
Mumbai, Maharashtra, India

Vandana Agarwal MD (Anesthesiology) FRCA
Professor
Department of Anesthesiology, Critical Care and Pain
Tata Memorial Hospital
Mumbai, Maharashtra, India

Vijaya Prakash Patil MD (Anesthesiology) Dip (Hosp Adminis)
Professor
Department of Anesthesiology, Critical Care and Pain
Tata Memorial Hospital
Homi Bhabha National Institute
Mumbai, Maharashtra, India



TATA MEMORIAL CENTRE

TATA MEMORIAL HOSPITAL

AND

ADVANCED CENTRE FOR TREATMENT, RESEARCH & EDUCATION IN CANCER

(A Grant-in-aid Institute under the Department of Atomic Energy, Govt. of India)

Dr (Prof) RA Badwe MS
Director

June 26, 2020

Foreword

'Objective Anesthesia Review' has had an enviable assessment in the minds of many and undoubtedly an affectionate patronage of the students and practitioners of art of anesthesia. The new edition has a remarkable focus on new equipments and at the same time has not forgotten the old adage "if you do not know the history, you are condemned to repeat it." References to the old equipments speaks of the evolution of process and safety of the delivery of anesthesia that has sound support of physics and physiology. As we master the techniques and overcome the most difficult problems, more complex problem confronts us and that is dealt with very effectively in the perioperative fluid management. The appeal of this book is that it not only prepares students for examination but equips them to practice safe anesthesia. This book exemplifies a good teacher, who is ruthless in pointing mistakes and following non-negotiable anesthesia processes; yet does not insist on one method, when there are many with identical outcomes.

I complement all those, who have contributed to this edition and I hope that every next edition is better than previous one.



RA Badwe MS
Director

Tata Memorial Hospital
Mumbai, Maharashtra, India

Preface

The first edition of *Objective Anesthesia Review: A Comprehensive Textbook for the Examinees* was published in 2009 with financial assistance from Tata Memorial Center, Mumbai, Maharashtra, in 2009. This was a culmination of lovingly nurtured fruit of labor, something we had been doing since 2004, when we started the first systematic and condensed teaching program called the 'Anesthesia Review Course' for the anesthesia postgraduates, who were about to face the examination. Like the course, this book discusses cases and anesthesia equipment, the examinees have to know, to get their license to practice. Much of the anesthesia equipment is of historical importance only, and comes out of that dreaded cupboard, only during the examinations. Many of our colleagues felt that this edition should get rid of these equipment from the book. However, the older equipment is a rich source of learning and many principles of physics and physiology are enshrined in it, so we have retained even the old equipment, in this edition as well.

In 2013, we were approached by Jaypee Brothers Medical Publishers (P) Ltd, New Delhi, India, saying they wanted to publish this book. This was great, since it meant that with Jaypee Brothers came their strong network of book sellers across the country, which meant many more students could lay their hand on the book. This indeed happened, and where ever we go, we find the anesthesia trainees reading the book. *Objective Anesthesia Review* has become really popular, thanks to you all, the hard working anesthesia trainees.

The older editions were in black and white, except for the color plates, since we wanted the cost of book to be kept low, so the students could afford it. This edition marks the transition to full color and very kindly the publishers have agreed to keep the cost affordable.

We have a new chapter on perioperative fluid therapy, an important topic, not only for examination but for day-to-day practice as well. We also wanted to change the chapter on Cardiopulmonary Resuscitation, however, the new CPR guidelines which were slated to published in January 2020, will now come out in November 2020. Please go through the new guidelines, when they are published, to update yourself.

Our colleagues in the department have worked hard year-over-year and contributed significantly to the book. They also endured our time away from clinical duties, so we could work on the book, ungrudgingly. Our sincere thanks to them. Ms Chetna Malhotra Vohra, an old friend by now (she is not old) who works with Jaypee Brothers Medical Publishers and Ms Rajul Jain, who was the Development Editor for the book, have been patient with us, acceding to our many demands. We are grateful for their patience and understanding. Big thanks to our families, who have been very supportive, without any nagging, while we worked on the book.

We hope the current and future examinees benefit from the book and do get back to us with your suggestions and criticisms, it will only make the next edition better! We wish you happy reading and success in your examination!

Atul Prabhakar Kulkarni
Jigeeshu Vasistha Divatia
Vijaya Prakash Patil

Acknowledgments

We wish to acknowledge the support of the premier institute, where we work, which has never stood in the way of giving knowledge to the others. Also our thanks to Dr Badwe (Director, Tata Memorial Center, Mumbai, Maharashtra) for the financial support for the first edition of *Objective Anesthesia Review: A Comprehensive Textbook for the Examinees*. Dr Sareen, our past head of the department and Dr Sharma, the director Academics for backing us in our every academic endeavor, thank you. Gratitude to Ms Chetna Malhotra Vohra (Associate Director—Content Strategy) and Dr Rajul Jain (Development Editor), from Jaypee Brothers Medical Publishers (P) Ltd, New Delhi, India, for their patience, perseverance and tolerance for the eccentric academics. We appreciate the help of all our colleague in the department gratefully and last but most important thanks to our spouses and children for their sufferance and forbearance.

Contents

Section 1: Case Discussions

1. Mitral Stenosis with Pulmonary Hypertension	3
<i>Vijaya Prakash Patil, Madhavi Shetmahajan</i>	
2. Ischemic Heart Disease	17
<i>Jigeeshu Vasistha Divatia, Jeson Rajan Doctor, Aparna Chatterjee</i>	
3. Anesthetic Considerations for Hypertensive Patient	35
<i>Vijaya Prakash Patil, Madhavi Shetmahajan, Sukhada Savarkar</i>	
4. Tetralogy of Fallot	45
<i>Vandana Agarwal, Reshma Ambulkar, Madhavi Desai</i>	
5. Anesthetic Considerations in a Patient with Patent Ductus Arteriosus	52
<i>Reshma Ambulkar, Vandana Agarwal, Malini Joshi</i>	
6. Permanent Pacemaker	58
<i>Sumitra Ganesh Bakshi, Vijaya Prakash Patil, Sheila Nainan Myatra</i>	
7. Peripheral Vascular Disease	69
<i>Parmanand Jain, Atul Prabhakar Kulkarni, Shilpushp Jagannath Bhosale</i>	
8. Pneumonectomy	75
<i>Priya Ranganathan, Madhavi Shetmahajan, Bhakti Trivedi</i>	
9. Bronchiectasis with Lung Abscess	86
<i>Priya Ranganathan, Amol Trimbakrao Kothekar, Swapnil Parab</i>	
10. Chronic Obstructive Pulmonary Disease	90
<i>Atul Prabhakar Kulkarni, Madhavi Desai, Aparna Chatterjee</i>	
11. Intercostal Drain	99
<i>Priya Ranganathan, Bhakti Trivedi, Sudivya Sharma, Madhavi Shetmahajan</i>	
12. Hypertensive Disorders in Pregnancy	104
<i>Sumitra Ganesh Bakshi, Reshma Ambulkar, Shilpushp Jagannath Bhosale</i>	
13. Pregnancy: Physiological Changes and Anemia	118
<i>Reshma Ambulkar, Sumitra Ganesh Bakshi, Madhavi Desai</i>	
14. Emergency Lower Segment Cesarean Section	128
<i>Sumitra Ganesh Bakshi, Reshma Ambulkar, Jeson Rajan Doctor</i>	
15. Nonobstetric Surgery in a Pregnant Patient	136
<i>Sumitra Ganesh Bakshi, Reshma Ambulkar, Malini Joshi</i>	
16. Amniotic Fluid Embolism	140
<i>Reshma Ambulkar, Sumitra Ganesh Bakshi, Amol Trimbakrao Kothekar</i>	
17. Obstetric Hemorrhage	143
<i>Reshma Ambulkar, Sumitra Ganesh Bakshi, Aparna Chatterjee</i>	

18. Hydrocephalus	151
<i>Sheila Nainan Myatra, Sumitra Ganesh Bakshi, Shilpushp Jagannath Bhosale</i>	
19. Meningomyelocele	160
<i>Sumitra Ganesh Bakshi, Sheila Nainan Myatra</i>	
20. Traumatic Brain Injury	165
<i>Atul Prabhakar Kulkarni, Malini Joshi, Sudivya Sharma</i>	
21. Managing Difficult Airway	174
<i>Vijaya Prakash Patil, Jeson Rajan Doctor, Debashree Lahiri</i>	
22. Burns	191
<i>Nayana Amin, Vijaya Prakash Patil, Bhakti Trivedi</i>	
23. Anesthesia for Cleft Lip and Palate Surgery	207
<i>Vandana Agarwal, Aparna Chatterjee</i>	
24. Geriatric Patient	214
<i>Amol Trimbakrao Kothekar, Raghu S Thota, Sukhada Savarkar</i>	
25. Transurethral Resection of Prostate	226
<i>Jigeeshu Vasistha Divatia, Shilpushp Jagannath Bhosale, Bindiya Salunke</i>	
26. Proximal Fracture Femur	236
<i>Jigeeshu Vasistha Divatia, Bhakti Trivedi, Debashree Lahiri</i>	
27. Cataract	244
<i>Sumitra Ganesh Bakshi, Debashree Lahiri, Sudivya Sharma</i>	
28. Morbid Obesity	250
<i>Vijaya Prakash Patil, Malini Joshi, Bindiya Salunke</i>	
29. Cirrhosis with Portal Hypertension	264
<i>Atul Prabhakar Kulkarni, Jigeeshu Vasistha Divatia, Sohan Lal Solanki</i>	
30. Laparoscopic and Robotic Cholecystectomy	280
<i>Priya Ranganathan, Debashree Lahiri</i>	
31. Colles' Fracture	288
<i>Parmanand Jain, Aparna Chatterjee</i>	
32. Kyphoscoliosis	293
<i>Parmanand Jain, Sukhada Savarkar, Atul Prabhakar Kulkarni</i>	
33. Large Thyroid Mass	301
<i>Madhavi Shetmahajan, Raghu S Thota, Bindiya Salunke</i>	
34. Diabetes Mellitus	313
<i>Madhavi Shetmahajan, Sohan Lal Solanki</i>	
35. Chronic Kidney Disease and Renal Transplant	332
<i>Vandana Agarwal, Sohan Lal Solanki</i>	
36. Tonsillectomy	346
<i>Sheila Nainan Myatra, Sumitra Ganesh Bakshi, Jeson Rajan Doctor</i>	
37. Circumcision	351
<i>Parmanand Jain, Sudivya Sharma</i>	
38. Acute Postoperative Pain	354
<i>Parmanand Jain, Sumitra Ganesh Bakshi, Malini Joshi</i>	

39. Pharmacokinetic Principles in Anesthesia	360
<i>Vandana Agarwal, Atul Prabhakar Kulkarni</i>	
40. Fluid Management in Major Abdominal Surgery	368
<i>Vandana Agarwal, Bindiya Salunke</i>	

Section 2: Anesthesia Equipment and Table Viva

41. Arterial Blood Gas Analysis	375
<i>Vijaya Prakash Patil, Jeson Rajan Doctor, Bindiya Salunke</i>	
42. Mechanical Ventilation	385
<i>Atul Prabhakar Kulkarni, Sohan Lal Solanki, Jigeeshu Vasistha Divatia</i>	
43. Pulmonary Function Tests	395
<i>Vijaya Prakash Patil, Madhavi Desai, Debashree Lahiri</i>	
44. Anesthesia Machine	406
<i>Nayana Amin, Vandana Agarwal</i>	
45. Vaporizers	432
<i>Priya Ranganathan, Madhavi Shetmahajan</i>	
46. Anesthesia Breathing Systems	451
<i>Sheila Nainan Myatra, Parmanand Jain</i>	
47. Endotracheal Tubes, Lung Isolation Devices and Combitube	462
<i>Atul Prabhakar Kulkarni, Aparna Chatterjee, Swapnil Parab</i>	
48. Cardiopulmonary Resuscitation	476
<i>Sheila Nainan Myatra, Bhakti Trivedi, Amol Trimbakrao Kothekar</i>	
49. Airways, Connectors, Laryngoscopes, and Non-rebreathing Valves	489
<i>Nayana Amin, Sudivya Sharma, Sukhada Savarkar</i>	
50. Electrocardiography	504
<i>Reshma Ambulkar, Swapnil Parab, Bindiya Salunke</i>	
51. X-ray Interpretation for the Budding Anesthesiologist	526
<i>Atul Prabhakar Kulkarni, Aparna Chatterjee, Debashree Lahiri</i>	
52. Supraglottic Airway Devices	534
<i>Vandana Agarwal, Sukhada Savarkar, Atul Prabhakar Kulkarni</i>	
53. Regional Anesthesia Instruments	549
<i>Parmanand Jain, Atul Prabhakar Kulkarni, Jeson Rajan Doctor</i>	
54. Nerve Locator and Peripheral Nerve Stimulator	554
<i>Nayana Amin, Sudivya Sharma</i>	
55. Oxygen Therapy Devices and Manual Resuscitator	566
<i>Nayana Amin, Vijaya Prakash Patil, Sohan Lal Solanki</i>	
56. Videolaryngoscopes	581
<i>Atul Prabhakar Kulkarni, Madhavi Desai, Shilpushp Jagannath Bhosale</i>	
57. Cardiopulmonary Exercise Test (CPET/CPEX/CPX)	592
<i>Vandana Agarwal, Vijaya Prakash Patil</i>	

SECTION

1

Case Discussions

- **Mitral Stenosis with Pulmonary Hypertension**
Vijaya Prakash Patil, Madhavi Shetmahajan
- **Ischemic Heart Disease**
Jigeeshu Vasistha Divatia, Jeson Rajan Doctor, Aparna Chatterjee
- **Anesthetic Considerations for Hypertensive Patient**
Vijaya Prakash Patil, Madhavi Shetmahajan, Sukhada Savarkar
- **Tetralogy of Fallot**
Vandana Agarwal, Reshma Ambulkar, Madhavi Desai
- **Anesthetic Considerations in a Patient with Patent Ductus Arteriosus**
Reshma Ambulkar, Vandana Agarwal, Malini Joshi
- **Permanent Pacemaker**
Sumitra Ganesh Bakshi, Vijaya Prakash Patil, Sheila Nainan Myatra
- **Peripheral Vascular Disease**
Parmanand Jain, Atul Prabhakar Kulkarni, Shilpushp Jagannath Bhosale
- **Pneumonectomy**
Priya Ranganathan, Madhavi Shetmahajan, Bhakti Trivedi
- **Bronchiectasis with Lung Abscess**
Priya Ranganathan, Amol Trimbakrao Kothekar, Swapnil Parab
- **Chronic Obstructive Pulmonary Disease**
Atul Prabhakar Kulkarni, Madhavi Desai, Aparna Chatterjee
- **Intercostal Drain**
Priya Ranganathan, Bhakti Trivedi, Sudivya Sharma, Madhavi Shetmahajan
- **Hypertensive Disorders in Pregnancy**
Sumitra Ganesh Bakshi, Reshma Ambulkar, Shilpushp Jagannath Bhosale
- **Pregnancy: Physiological Changes and Anemia**
Reshma Ambulkar, Sumitra Ganesh Bakshi, Madhavi Desai
- **Emergency Lower Segment Cesarean Section**
Sumitra Ganesh Bakshi, Reshma Ambulkar, Jeson Rajan Doctor
- **Nonobstetric Surgery in a Pregnant Patient**
Sumitra Ganesh Bakshi, Reshma Ambulkar, Malini Joshi
- **Amniotic Fluid Embolism**
Reshma Ambulkar, Sumitra Ganesh Bakshi, Amol Trimbakrao Kothekar
- **Obstetric Hemorrhage**
Reshma Ambulkar, Sumitra Ganesh Bakshi, Aparna Chatterjee
- **Hydrocephalus**
Sheila Nainan Myatra, Sumitra Ganesh Bakshi, Shilpushp Jagannath Bhosale
- **Meningocele**
Sumitra Ganesh Bakshi, Sheila Nainan Myatra
- **Traumatic Brain Injury**
Atul Prabhakar Kulkarni, Malini Joshi, Sudivya Sharma
- **Managing Difficult Airway**
Vijaya Prakash Patil, Jeson Rajan Doctor, Debashree Lahiri
- **Burns**
Nayana Amin, Vijaya Prakash Patil, Bhakti Trivedi
- **Anesthesia for Cleft Lip and Palate Surgery**
Vandana Agarwal, Aparna Chatterjee
- **Geriatric Patient**
Amol Trimbakrao Kothekar, Raghu S Thota, Sukhada Savarkar
- **Transurethral Resection of Prostate**
Jigeeshu Vasistha Divatia, Shilpushp Jagannath Bhosale, Bindiya Salunke
- **Proximal Fracture Femur**
Jigeeshu Vasistha Divatia, Bhakti Trivedi, Debashree Lahiri
- **Cataract**
Sumitra Ganesh Bakshi, Debashree Lahiri, Sudivya Sharma
- **Morbid Obesity**
Vijaya Prakash Patil, Malini Joshi, Bindiya Salunke
- **Cirrhosis with Portal Hypertension**
Atul Prabhakar Kulkarni, Jigeeshu Vasistha Divatia, Sohan Lal Solanki
- **Laparoscopic and Robotic Cholecystectomy**
Priya Ranganathan, Debashree Lahiri
- **Colles' Fracture**
Parmanand Jain, Aparna Chatterjee
- **Kyphoscoliosis**
Parmanand Jain, Sukhada Savarkar, Atul Prabhakar Kulkarni
- **Large Thyroid Mass**
Madhavi Shetmahajan, Raghu S Thota, Bindiya Salunke
- **Diabetes Mellitus**
Madhavi Shetmahajan, Sohan Lal Solanki
- **Chronic Kidney Disease and Renal Transplant**
Vandana Agarwal, Sohan Lal Solanki
- **Tonsillectomy**
Sheila Nainan Myatra, Sumitra Ganesh Bakshi, Jeson Rajan Doctor
- **Circumcision**
Parmanand Jain, Sudivya Sharma
- **Acute Postoperative Pain**
Parmanand Jain, Sumitra Ganesh Bakshi, Malini Joshi
- **Pharmacokinetic Principles in Anesthesia**
Vandana Agarwal, Atul Prabhakar Kulkarni
- **Fluid Management in Major Abdominal Surgery**
Vandana Agarwal, Bindiya Salunke

Mitral Stenosis with Pulmonary Hypertension

Vijaya Prakash Patil, Madhavi Shetmahajan

A 42-year-old lady, with a history of rheumatic mitral stenosis, presents with a large pelvic mass suspected to be an ovarian cyst. She is scheduled for excision of the mass. She gives a history of balloon mitral valvotomy 13 years ago. She also gives a history of normal pregnancy 15 years ago. She can climb two flights of stairs without difficulty but feels breathless beyond this.

Q. What are the causes of mitral stenosis (MS)?

Ans. Mitral stenosis refers to a decrease in the mitral valve area. The most common cause of MS is rheumatic heart disease (RHD), although >50% of these patients do not give a history suggestive of rheumatic fever in the past. Rheumatic fever leads to cardiac inflammation causing pancarditis which heals with scarring, affecting valves. In the acute phase, rheumatic fever may cause mitral regurgitation. MS develops few years later and symptoms develop after many more years when the mitral valve area reduces significantly. The stenosis is characterized by fusion, fibrosis, thickening, and calcification of the leaflets, and thickening, fusion, and shortening of the chordae tendineae.

Other causes of MS include congenital MS, degenerative MS due to mitral annular calcification, systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), malignant carcinoid, mucopolysaccharidoses (of the Hunter-Hurler phenotype), Fabry disease, Whipple disease, and methysergide therapy.

Q. Describe the pathophysiology of MS.

Ans. The normal mitral valve area is 4–6 cm². In very early diastole, there is a small pressure gradient between left atrium (LA) and left ventricle (LV), which rapidly equilibrates in the phase of diastasis followed by a phase of atrial contraction.

As the mitral orifice narrows in MS, it obstructs free flow of blood from LA to LV during diastole, leading to the development of a pressure gradient between two chambers and an increase in LA and pulmonary venous pressure. This gradient is increased by increases in the heart rate (reducing diastolic time) or cardiac output (CO) (increasing flow of blood across the mitral valve). As obstruction becomes

more and more severe, the time required for left ventricular filling increases and left atrial contraction (kick) becomes necessary to fill the ventricle. As the mitral valve area goes below 1 cm², left atrial pressure (LAP) increases to about 25 mm Hg (normal LA pressure is about 5 mm Hg). Since there are no valves between pulmonary circulation and LA, increased LAP is transmitted to pulmonary circulation causing pulmonary hypertension. Also, this constant pressure overload of the LA leads to the increase in left atrial size, which then becomes more prone to develop atrial fibrillation (AF) and atrial thrombus formation.

As stenosis worsens, flow restriction also limits left ventricular filling and thus LV preload. In addition, there is increased afterload due to reflex vasoconstriction in response to low CO. Both these factors result in decreased LV function, leading to further reduction in CO and thus may mimic left ventricular failure.

In its early stage, the pulmonary hypertension is often described as “passive” and reversible, as elevation of pulmonary arterial pressure is caused solely by passive backward transmission of the elevated LAP. Over a period of time, this leads to pulmonary artery and arteriole vasoconstriction called reactive pulmonary hypertension. Over a longer period, the muscular layer of pulmonary arterioles hypertrophies in response to elevated pulmonary vascular pressures, resulting in irreversible pulmonary hypertension. Increased pulmonary artery pressure (PAP) also leads to pressure overload for right ventricle (RV) which hypertrophies and ultimately dilates and fails. Thus, in MS, two “stenoses” develop: early at the mitral valve and later in the arterioles of the lung. Workup of the patient with MS should include an assessment of both these obstructions.

Q. Describe the natural history of MS.*Ans.*

- Mitral stenosis is a continuous, progressive, lifelong disease.
- It is generally slow but often with a variable clinical course, with some patients showing little or no clinical progression over prolonged periods and others manifesting a more rapid course.
- Onset of symptoms to disability: 10 years.
- Incidence of AF: 30–40%.
- Causes of death:
 - Congestive heart failure (CHF)
 - Systemic embolism
 - Pulmonary embolism
 - Infection.

Q. What are the signs and symptoms of MS?

Ans. History and physical examination should focus on the patient's effort, tolerance, current or past evidence of heart failure, and arrhythmias. In mild disease, the patient may be entirely asymptomatic. As the disease progresses, the patient will start exhibiting signs and symptoms according to severity of stenosis and underlying cardiac changes. Symptoms can be classified according to the causation.

- *Due to decreased CO:*
 - Low effort tolerance
 - Typical “mitral” facies—mild cyanosis of lips and cheeks and malar prominence
 - Easy fatigability
 - Syncope
- *Due to increased LAP:*
 - Pulmonary congestion giving rise to dyspnea and orthopnea
 - Hemoptysis as pulmonary venous hypertension results in rupture of anastomoses between bronchial veins
 - Pulmonary edema
- *Due to LA enlargement:*
 - Ortner's syndrome (hoarseness due to compression of the left recurrent laryngeal nerve by LA)
 - AF and thromboembolism
- *Due to pulmonary hypertension, RV hypertrophy, and RV failure:*
 - Chest pain due to RV ischemia in severe pulmonary hypertension
 - Raised jugular venous pressure (JVP)
 - Right parasternal heave
 - Tricuspid murmur
 - Hepatomegaly
 - Ascites and edema.

Q. What are the causes of hemoptysis?*Ans.**Airway diseases:*

- Airway trauma
- Acute or chronic bronchitis
- Bronchiectasis
- Foreign body in airway
- Lung cancer
- Tracheal or bronchial tumors

Parenchymal disease:

- Tuberculosis
- Pneumonia
- Lung abscess
- Mycetoma
- Goodpasture's syndrome
- Idiopathic pulmonary hemosiderosis
- Pulmonary contusion

Vascular causes:

- Pulmonary embolism
- Pulmonary hypertension:
 - LV failure
 - MS
- Pulmonary vascular malformation.

Q. How will you grade the severity of MS?

Ans. The grades of severity of MS are given in **Table 1**.

TABLE 1: Grades of mitral stenosis.

Severity	Moderate mitral stenosis (MVA) (cm ²)	End diastolic pressure gradient, mm Hg (in sinus rhythm)	Pulmonary artery pressure (PAP) (mm Hg)	Symptoms
Mild	>1.5	<5	Normal <30	Usually absent
Moderate	1.0–1.5	5–10	30–50	NYHA Class II
Severe	<1.0	>10	>5	NYHA Classes III and IV

(NYHA: New York Heart Association).

Q. What are the cardiac findings on examination in MS?

Ans. Loud S1: The transmitral gradient holds the mitral valve open almost to the end of diastole, so that at the beginning of ventricular systole, the mitral valve leaflets move along a long distance for closure. In a badly diseased valve, S1 may become soft because it neither opens nor closes well. This manifests as tapping apex beat on palpation.

Loud P₂: The pulmonic component of the second sound (A₂P₂) will be loud in presence of pulmonary hypertension.

Opening snap: The distance from S2 to the opening snap is a good indicator of MS severity. The more severe the stenosis,

the higher will be LAP and sooner will the mitral valve open. An S2-opening snap interval <0.08 seconds usually indicates severe disease.

Murmur: Diastolic murmur (low-pitched) at the apex with a presystolic accentuation in a patient with sinus rhythm. A diastolic thrill may be palpated in the left lateral position.

Diastolic murmur (high-pitched), also called Graham Steel murmur, is due to the pulmonary regurgitation secondary to pulmonary hypertension. It is heard at the left sternal border in the second intercostal space with inspiration.

Right parasternal heave: This is due to enlarged RV and tricuspid murmur in patients with pulmonary hypertension and right ventricular dilatation.

Q. What is the differential diagnosis for a mid-diastolic murmur?

Ans.

- Mitral stenosis
- Tricuspid stenosis
- Atrial myxoma
- Ball-valve thrombus in LA
- Increased flow across the tricuspid valve in atrial septal defect (ASD)
- MR with increased flow through the mitral valve during diastole
- Austin Flint murmur (in severe AR).

Q. Enumerate electrocardiogram (ECG) findings in a patient with MS.

Ans.

- Left atrial hypertrophy:
 - *Causes P-mitrale:* Bifid appearance of P-wave in leads II, III, and aVF
 - The second half of the P-wave is negative in V1
- AF
- *Right ventricular hypertrophy:*
 - Dominant R wave in V1 and V2
- Right axis deviation
- For patients on digoxin, digitalis effects maybe seen (ST segment depression with a characteristic sagging appearance, decreased T-wave amplitude, shortened QT interval, prominent U-waves).

Q. Enumerate X-ray findings in a patient with MS.

Ans.

- Usually normal or slightly enlarged cardiothoracic ratio (normal 0.5)
- Straightening of left heart border due to enlarged LA appendage
- Small aortic knuckle due to decreased CO
- Enlarged pulmonary conus in pulmonary hypertension

CHAPTER 1: Mitral Stenosis with Pulmonary Hypertension

- Moustache or antler sign—prominence of upper lobe veins
- Double density in midportion of cardiac silhouette due to LA enlargement
- Elevation of the left main stem bronchus due to enlarged LA
- Left lower lobe collapse due to compression of the left lower lobe bronchus
- Miliary shadows of pulmonary hemosiderosis due to multiple pulmonary hemorrhages
- Calcification of the mitral valve
- Indentation of esophagus on lateral X-ray with barium swallow due to enlarged LA.

Q. Describe the anatomy of the mitral valve.

Ans.

Leaflet:

- Small anterior leaflet inserts into about one-third of the annulus
- Large posterior leaflet inserts into about two-thirds of the annulus

Chordae:

- Primary attachment to free margin
- Secondary and tertiary attachment away from free margin

Papillary muscles:

- Anterolateral and posteromedial supplying both leaflets.

Q. What causes presystolic accentuation of the murmur?

Ans. This would be heard only if the patient is in sinus rhythm. As the mitral valve area goes on decreasing, the pressure gradient between LA and LV goes on increasing. In severe MS, LV preload becomes more and more dependent on atrial contraction. During atrial contraction, there is increased flow from the LA to the LV through the stenotic valve which gives rise to presystolic accentuation.

Q. How do you diagnose rheumatic fever? What are Duckett Jones criteria?

Ans. Duckett Jones criteria: For diagnosis of rheumatic fever, there should be either two major or one major + two minor criteria present:

- *Major:* Carditis, Sydenham's chorea, subcutaneous nodules, erythema marginatum, arthritis.
- *Minor:* Past history of RHD, fever, arthralgia, prolonged PR on ECG, increased erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP), leukocytosis.

Q. What is Ortner's syndrome?

Ans. When a patient develops hoarseness of voice from compression of the recurrent laryngeal nerve from an enlarged LA, it is called Ortner's syndrome. It was first described in 1897 by N Ortner, an Austrian physician.

Q. What is Lutembacher's syndrome?

Ans. It is a combination of the presence of MS with ASD (of the ostium secundum type) with a left-to-right shunt.

Q. Explain the basic principles of echocardiography. What do you understand by M-mode/B-mode and Doppler?

Ans. Echocardiographic systems emit a high-frequency sound (>20,000 Hz) into the tissues. These waves enter the tissue and are then reflected back from the various tissues based on acoustic impedance of the tissues. Since the speed of sound in tissues is known (~1540 m/sec), a standard ultrasound imaging system can wait for a given time for the transmitted pulse to travel to a target (time $\times$) and then back (time $2\times$); the given target will be received and recorded. These reflected ultrasound waves received by the transducer are then processed by the ultrasound machine to create an image. The best ultrasound images are made when the target is perpendicular to the sound waves.

In the *M-mode technique*, all the ultrasonic pulses are transmitted along the same axis and different parts of the heart are studied by changing the direction of the beam manually. An M-mode echocardiogram is not a "picture" of the heart, but rather a diagram that shows how the positions of its structures change during the course of the cardiac cycle. It is a method for studying opening and closing of structure like a heart valve.

B-mode or two-dimensional (2D) echocardiography allows a plane of tissue (both depth and width) to be imaged in real time. Thus, the anatomic relationships between various structures are easier to appreciate than with the M-mode echocardiographic images.

Doppler echocardiography is a method for detecting the direction and velocity of moving blood within the heart or vessel. It measures the relative change in the returned ultrasound frequency when compared to the transmitted frequency. Depending on this, the Doppler echocardiographic systems measure flow direction, velocity, and turbulence of the flow. This helps in assessment of valve area, regurgitation, transvalvular pressure gradient, presence of septal defects, assessment of diastolic function, and calculation of CO.

Q. What are echocardiographic findings in a patient with MS?

Ans.

M-mode:

- Thickened mitral valve leaflets
- Decreased E-F slope of the anterior mitral valve leaflet to usually <30 mm/sec
- Reduced anterior motion of the posterior mitral valve leaflet
- Decreased A-wave of the mitral valve leaflet

- Reduced mitral valve leaflet excursion (D-E excursion)
- Abnormal septal wall motion (paradoxical or flat) due to right ventricular volume and/or pressure overload
- Left atrial enlargement
- Pulmonary hypertension.

2D echocardiography, also called B-mode:

- Thickened mitral valve leaflets, especially at the leaflets and chordae
- Left atrial enlargement
- Left atrial thrombus (if present)
- Normal LV
- Right ventricular and right atrial enlargement in pulmonary hypertension.

Q. What is the role of infective endocarditis prophylaxis in MS?

Ans. The current American Heart Association (AHA) recommendations no longer suggest infective endocarditis prophylaxis for patients with RHD. Only the high-risk group should receive prophylaxis against infective endocarditis. The high-risk groups are as follows:

- Patients with a prosthetic heart valve or prosthetic material used for valve repair
- Patients with a past history of infective endocarditis
- Patients with cardiac valvulopathy after cardiac transplantation
- Specific patients with congenital heart disease.

Q. What are the treatment strategies available for a patient with MS?

Ans.

Medical management: It mainly consists of:

- Diuretics if congestive symptoms present
- *In the presence of AF:* Anticoagulants such as warfarin and drugs for ventricular rate control such as calcium channel blockers/ β -blockers.

Surgical management: Surgical options mainly consist of balloon mitral valvuloplasty, surgical valvulotomy or repair, or mitral valve replacement. Patients with moderate or severe MS (mitral valve area < 1.5 cm²) and suitable valve are good candidates for percutaneous balloon mitral valvuloplasty. The major contraindications to balloon mitral valvuloplasty are the presence of thrombus in the LA or its appendage, moderate-to-severe mitral regurgitation, and an unfavorable valve morphology (mainly badly calcified or scarred valves). In this group, options are either open mitral valvulotomy or valve replacement where the valve cannot be repaired.

Q. On physical examination, she is comfortable and in good general condition. Her blood pressure is 130/70 mm Hg and pulse rate is 80 beats/min (regular). There is no jugular venous distention, the lungs are normal, and

the point of maximal impulse is in the fifth intercostal space in the midclavicular line. The first heart sound is accentuated; an opening snap and a grade 2 diastolic murmur with presystolic accentuation can be heard at apex. There is no hepatomegaly or peripheral edema. What investigations would you ask for this patient?

Ans.

- Complete blood count
- Blood grouping and cross matching
- Liver function test
- Renal function test and electrolytes to see the effect of diuretics and digoxin if the patient is receiving any
- ECG
- Chest X-ray
- Echocardiogram—mitral valve area, presence of mitral regurgitation, LA clot, pulmonary pressures.

Q. The ECG is normal. The X-ray chest shows a normal-sized heart, an enlarged LA, no calcification in the region of the mitral valve, and normal lung fields. Echocardiogram shows moderate MS (MVA 1.2 cm²) with evidence of mild pulmonary artery hypertension (PAH 38 mm Hg). How would you anesthetize this patient? What premedication would you prescribe?

Ans. Sedatives and anxiolytics: Prevention of tachycardia is very important as it worsens the left ventricular filling by decreasing diastolic time. Elimination of anxiety avoids tachycardia, but excessive sedation may lead to respiratory depression and hypercarbia and acidosis, resulting in elevated pulmonary vascular resistance (PVR). So, sedative premedication such as benzodiazepines must be used judiciously in titrated doses.

Antibiotic prophylaxis: This patient would need antibiotic prophylaxis for her surgery which would include good gram-positive and gram-negative cover. The drug can be given as per institutional protocol.

Deep venous thrombosis (DVT) prophylaxis: Since this patient is active with normal lifestyle, chances of preoperative DVT are less. Patients with severe MS with restricted lifestyle should be screened for DVT preoperatively and put on prophylaxis in perioperative period. If epidural catheter insertion is planned confirm that there is a gap of minimum 12 hours between LMWH and epidural catheter placement as per ASRA guidelines.

Q. What medications these patients are likely to be taking and would you continue these drugs?

Ans. As we have seen earlier, these patients are mostly treated with diuretics to relieve congestive symptoms—digoxin in case of AF and/or failure, β -blockers or calcium channel blockers for rate control, and anticoagulants in case of AF.

If the patient is on diuretics, care should be taken to evaluate the fluid status carefully, as hypovolemia in a patient with MS may give rise to a precipitous fall in BP during induction of anesthesia. Check electrolytes on the day of surgery and correct any existing dyselectrolytemia. It is preferable to withhold diuretics on the night before surgery, especially if massive fluid shifts or blood loss is expected during surgery. Drugs to control AF and rate should be continued in the perioperative period; monitor serum potassium in patients receiving digoxin and diuretics. Patients with AF on warfarin should be converted to heparin (infusion) preoperatively. Heparin infusion should be titrated to maintain activated partial thromboplastin time (APTT) 1.5–2 times normal and continued in the postoperative period.

Q. Describe the principles of anesthetic management.

Ans.

- Establish intra-arterial BP and central venous pressure (CVP) monitoring. CVP monitoring is of much less value in patients with right ventricular dysfunction.
- Avoid tachycardia.
- Judicious fluid therapy to maintain preload—hypovolemia may lead to a marked drop in CO and hypervolemia can lead to pulmonary edema.
- Avoid fall in systemic vascular resistance (SVR) or vasodilators, especially in tight MS.
- *Avoid increases in PVR:* Avoid hypoxia, hypercarbia, nitrous oxide.
- Maintain sinus rhythm; be prepared to cardiovert (synchronized cardioversion) if the patient gets AF. Volatile anesthetics may lead to junctional rhythm with loss of atrial kick.

Q. How would you anesthetize this patient?

Ans. There is no ideal technique or drug. Any technique can be used as long as the hemodynamic goals are met:

- Optimize fluid status before induction and establish invasive pressure monitoring.
- Use a generous amount of opioids to abolish hemodynamic response to intubation. It will also decrease the requirement of induction agents. Remifentanyl, alfentanil, or fentanyl can be used. Since Alfentanil and Remifentanyl are not available in India, Fentanyl is the drug of choice. Patients with moderate-to-severe MS generally have slow circulation that prolongs arm–brain circulation time. Dilute concentrations of induction agents should be given slowly in titrated doses till anesthesia is induced to prevent precipitous hypotension. Etomidate is the best agent for hemodynamic stability, but midazolam or thiopentone can also be used. Propofol should be avoided as it can lead to precipitous hypotension.

Vecuronium, atracurium, or rocuronium can be used as they have the least effect on heart rate. Pancuronium and D-tubocurarine should be avoided due to their tachycardic response.

- Use titrated doses of vasopressors such as phenylephrine or noradrenaline to maintain BP and short acting β -blockers such as esmolol or metoprolol or calcium channel blockers such as diltiazem to control heart rate.

Q. How will you maintain anesthesia?

Ans. Volatile anesthetics such as isoflurane, desflurane, or sevoflurane can be administered. Halothane is best avoided due to its arrhythmogenic potential and high incidence of junctional rhythm, which can be disastrous in a patient with MS. In patients with pulmonary artery hypertension, nitrous oxide is best avoided due to its effects on pulmonary resistance. Monitoring of CVP may help in fluid therapy in the absence of right ventricular dysfunction.

Q. What is the role of regional anesthesia?

Ans. In valvular heart disease, concern is with moderate-to-severe grades of aortic or MS and neuraxial blockade. Epidural anesthesia can be used as a sole anesthesia technique in patients with mild-to-moderate MS. A number of case reports have been published and reviewed in support of this fact. Care should be taken to optimize fluid status and achieve sensory level with titrated doses of local anesthetic agents until it is adequate for surgery. The basic principles of anesthesia remain the same, i.e., minimize drop in afterload reduction, maintenance of sinus rhythm, careful volume management, and avoidance of tachycardia. Epidural analgesia with opioids can be safely considered as a supplement to general anesthesia (GA) for other patients. While inserting the epidural catheter, use of adrenaline in the epidural test dose should be avoided.

Q. What precautions would you take to prevent increase in PVR intraoperatively?

Ans.

Avoid:

- Hypoxia
- Hypercarbia
- Acidosis
- Lighter planes of anesthesia
- Hypervolemia.

Q. Describe management of perioperative AF.

Ans. In MS, due to the pressure gradient between the LA and LV, ventricular filling is dependent on atrial systole and is deleteriously affected in AF. In addition, AF causes stasis of blood in the LA, increasing the risk of thrombosis and systemic thromboembolism.

Control of AF with rapid ventricular response should be considered with any of the following agents:

- *Metoprolol*: 5 mg bolus and repeat if needed

- *Esmolol*: Bolus of 500 μ g/kg IV over 1 minute; may repeat in 5 minutes
- *Diltiazem*: 10–15 mg IV in slow titrated doses; may repeat in 15 minutes
- *Digoxin*: 0.25–0.5 mg IV; then 0.25 mg IV every 4–6 hours to a maximum of 1 mg
- *Amiodarone*: 150 mg IV bolus over 15 minutes, followed by 60 mg/h for 6 hours, then 30 mg/h with repeat boluses of 150 mg IV as needed up to a maximum of 2.0 g/day.

All antiarrhythmic agents have negative inotropic action and hence if the patient is hemodynamically unstable, one should consider immediate cardioversion (synchronized 100 J, 200 J, 300 J, then 360 J monophasic, or biphasic equivalent, with prior sedation).

In case of acute-onset AF there is no need to anticoagulate the patient but if AF is present for >48 hours, the patient should be anticoagulated before trying any form of cardioversion (electrical or pharmacological).

Anticoagulation is necessary in patients who are unable to maintain normal sinus rhythm.

Anticoagulation may also be beneficial for patients with normal sinus rhythm with a prior embolic event or a left atrial dimension >55 mm Hg noted by echocardiography.

Q. What are the anatomical considerations for percutaneous mitral commissurotomy (PMC) or balloon mitral valvotomy (BMV)?

Ans. Wilkins mitral valve score predicts outcome after PMC.

Wilkins Mitral Valve Score (On Transthoracic 2D Echocardiography)

Leaflet mobility:

- Highly mobile valve with only leaflet tips restricted
- Leaflet mid- and base portions have normal mobility
- Valve continues to move forward in diastole, mainly from the base
- No or minimal forward movement of the leaflets in diastole.

Leaflet thickness:

- Leaflets near normal in thickness (4–5 mm)
- Mid-leaflets normal, considerable thickening of margins (5–8 mm)
- Thickening extending through the entire leaflet (5–8 mm)
- Considerable thickening of all leaflet tissue (>8–10 mm).

Leaflet calcification:

- A single area of increased echo brightness
- Scattered areas of brightness confined to leaflet margins
- Brightness extending into the midportion of the leaflets
- Extensive brightness throughout much of the leaflet tissue.

Subvalvular thickening:

- Minimal thickening just below the mitral leaflet

- Thickening of chordal structures extending to one-third of the chordal length
- Thickening extending to the distal third of the chords
- Extensive thickening and shortening of all chordal structures extending down to the papillary muscles.

Each category is scored 0–4, for a total score ranging from 0 to 16. Generally, a score of <9 predicts optimal result while a score of >11 signifies severe disease and a suboptimal result after percutaneous mitral valvulotomy. The score does not perform well when the scores are between 9 and 11.

Q. What types of valves are available? Discuss briefly about each valve.

Ans.

Bioprosthetic valves—developed from animal tissues, mainly porcine or bovine:

- They have low incidence of thromboembolism.
- Their average durability is 10–15 years.
- They are chosen in females desiring to become pregnant.
- Glutaraldehyde-preserved stented porcine tissue valves are the most common.
- When bioprosthetic valve degeneration is detected, repair should be completed before the onset of symptoms due to the possibility of rupture and embolism.

Mechanical valves:

- They are durable with valve life up to 20 years.
- They require anticoagulation [international normalized ratio (INR) 2.5–3.0].
- They are usually indicated in young patients or in patients with chronic AF.
- *Three types of mechanical valves are:*
 1. Caged-ball (Starr-Edwards)
 2. Tilting disk (Medtronic, omniscience)
 3. Bileaflet (St. Jude, carbomedics).

Q. What are the indications for percutaneous BMV?

Ans.

- *Class I*—symptomatic patients (NYHA II, III, IV), moderate or severe MS (valve area < 1.5) and valve morphology favorable to balloon valvotomy in the absence of LA thrombus or moderate-to-severe MR.
- *Class IIb*—asymptomatic patients with moderate or severe MS (valve area < 1.5) and valve morphology favorable to balloon valvotomy in the absence of LA thrombus or moderate-to-severe MR.

Q. What are the indications for mitral valve repair?

Ans.

- Symptomatic patients (NYHA III, IV), moderate or severe MS (valve area < 1.5) and valve morphology favorable for repair if balloon valvotomy is not available.
- Symptomatic patients (NYHA III, IV), moderate or severe MS (valve area < 1.5) and valve morphology

favorable to repair if LA thrombus is present despite anticoagulation.

- Presence of nonpliable or calcified valve.
- Recurrent episodes of embolic events in spite of adequate anticoagulation.

Q. What are the indications for mitral valve replacement?

Ans.

- Symptomatic patients (NYHA II, III, IV), moderate or severe MS (valve area < 1.5) who are not candidates for percutaneous balloon valvotomy or valve repair.
- Patients with severe MS (valve area < 1) and severe pulmonary hypertension (pulmonary systolic pressures > 60–80 mm Hg) who are not candidates for percutaneous balloon valvotomy or valve repair.

Q. What are the indications for anticoagulation?

Ans.

- Patients with AF (class Ib)
- Prior embolic event even in sinus rhythm (class Ib)
- Thrombus in LA (class Ib)
- Severe MS and left atrial dimensions 55 mm on echocardiogram (class II b).

■ ANESTHETIC CONSIDERATIONS FOR A PREGNANT PATIENT WITH MITRAL STENOSIS AND PULMONARY HYPERTENSION

Q. Why does pregnancy aggravate the symptoms of MS?

The main cardiovascular changes in pregnancy which worsen features of MS are as follows:

- Increase in blood volume by 30–50% starting at the end of the first trimester to peak at 20–24 weeks. This increases pulmonary capillary hydrostatic pressure, thereby increasing the risk of pulmonary edema.
- Decrease in SVR.
- Increase in heart rate by 10–20 beats/min—reduces diastolic filling time of LV.
- CO increases by 30–50% after the fifth month. CO returns to normal within 3 days of delivery. Since a transvalvular gradient increases by the square of CO, the transvalvular gradient increases significantly. This also raises LA pressure substantially to give rise to symptoms. During pregnancy, the patient's symptomatic status will generally increase by 1 New York Heart Association (NYHA) class.
- During labor and delivery, there is sympathetic stimulation causing tachycardia and further increase in CO. Also, there is sudden rise in venous return to the heart due to autotransfusion from the placental circulation and IVC decompression. This may lead to decompensation.
- Enlarged atrial dimension predisposes to atrial arrhythmias including AF.

- Pregnancy also induces changes in hemostasis which contribute to increased coagulability and thromboembolic risk. The thrombosis risk is highest in the first 6 weeks after delivery but remains elevated for 12 weeks.

Q. How is pregnancy managed in a patient with MS?

Ans. Pregnancy in a patient with valvular heart disease needs a multidisciplinary approach with involvement of the obstetrician, cardiologist, and anesthesiologist with detailed counseling of the patient and her family.

Pregnancy should be deferred till a symptomatic MS is treated as there is worsening of symptoms and high risk of maternal and fetal mortality and morbidity. Patients with MVA $< 1.0 \text{ cm}^2$ fare better with PMC than medical management if pregnancy is planned. The decision to perform PMC before conception in patients with moderate MS should be on the basis of their valve area, symptoms, and exercise tolerance. Patients with MVA $> 1.5 \text{ cm}^2$ usually have a favorable pregnancy outcome. Unfortunately, in many patients in the developing countries, MS is first diagnosed during pregnancy as the physiological stress of pregnancy worsens symptoms.

Dyspnea in pregnancy could be due to various reasons but onset in the second trimester (especially between 3rd and 5th months) corresponding to a rise in CO should be evaluated further to detect valvular disease.

Mortality in pregnant women with minimal symptoms is $< 1\%$. The risk of maternal cardiac complications is high in

cases of severe stenotic valvular disease, i.e., MVA $< 1.5 \text{ cm}^2$ and severe pulmonary hypertension (systolic PAP $> 75\%$ of systemic pressure). The risk is increased in women with a history of cardiac events, arrhythmias, LV dysfunction [ejection fraction (EF) $< 40\%$] or who are in NYHA classes II, III, and IV.

Fetal risk is also high in stenotic heart valve disease due to growth retardation, preterm delivery, and still birth.

Echocardiographic examination should be performed in all pregnant patients presenting with dyspnea or heart murmur (except the soft systolic murmurs) or those who have a prosthetic valve. Valve stenosis should be quantified using valve area measurement. Though transvalvular gradients are not reliable markers of severity in pregnancy (because of increased CO), they have a prognostic value. The patient should also be evaluated for pulmonary hypertension and LV function. Chest X-rays should be limited to when absolutely required with appropriate shielding of the abdomen. CT is contraindicated because of the radiation risk, but MRI can be performed during pregnancy. Cardiac catheterization is restricted to the interventional procedures with abdominal shielding.

Medical therapy: Fetal side-effects should be considered when prescribing drugs. Medical management is advocated in patients with mild-to-moderate symptoms before pregnancy where the aim is to reduce overload and avoid tachycardia. Fetal side-effects should be considered when prescribing drugs (**Table 2**).

TABLE 2: Drug therapy and their implications.

Drugs	Indications	Fetal side-effects
Furosemide	To decrease congestion associated with valvular heart disease levels	Increased urinary sodium and potassium levels
β -blockers	Hypertension, supraventricular arrhythmias, to control heart rate in women with clinically significant mitral stenosis	Possible decreased heart rate, possible lower birth weight
Warfarin	For anticoagulation of mechanical heart valves, valvular heart disease with associated atrial fibrillation during 12–36 weeks of pregnancy	Hemorrhage, developmental abnormalities when used between 6 and 12 weeks of gestation
Unfractionated heparin	For anticoagulation of mechanical heart valves, valvular heart disease with associated atrial fibrillation during 6–12 weeks and after 36 weeks of pregnancy	Hemorrhage, no congenital defects
Low-molecular-weight heparin	Use during pregnancy is controversial	Hemorrhage
Aspirin	Low-dose aspirin occasionally used as an adjunct in patients with previous embolic events or prosthetic-valve thrombosis	Hemorrhage, prolongation of labor, low birth weight (when taken in high doses)
Digoxin	For suppression of supraventricular arrhythmias	No major adverse effects
Adenosine	For immediate conversion of supraventricular arrhythmias	No major adverse effects
Amiodarone	May be used to suppress atrial or ventricular arrhythmias in high-risk patients	Hypothyroidism, intrauterine growth retardation, premature birth. Rarely used during pregnancy because of side effects

Treatment includes bed rest, salt restriction, β -blockers with β_1 selectivity (interfere less with β_2 -mediated uterine relaxation) and diuretics if appropriate. β -blockers are safe but may induce neonatal bradycardia and possible growth retardation. Higher doses of β -blockers are required in pregnancy due to increased heart rate in pregnancy. Metoprolol is better than atenolol due to reports of fetal growth retardation with the latter (β -agonist agents are contraindicated).

Atrial fibrillation should be treated with cardioversion only when it is acute in onset or after intervention. (The cause of AF in MS is an enlarged LA. So, there is a high chance of reversion to fibrillation prior to definitive treatment.) Digoxin, β -blockers, and calcium channel blockers are used for rate control. Verapamil is preferred over diltiazem due to report of fetal side effects. Anticoagulation therapy is needed to avoid thromboembolism in case of chronic AF. Anticoagulant therapy should be also considered in patients with left atrial enlargement and those with low CO. Diuretics can impair fetal perfusion and should be used at the lowest dose possible. Quinidine and procainamide can be given for atrial and ventricular arrhythmias as they have a good safety profile in pregnancy.

During pregnancy, clinical and echocardiographic follow-up should be performed at the 3rd and 5th months and every month thereafter in pregnant patients with severe valve stenosis and whenever there is worsening of symptoms.

In the case of dyspnea or PAH not responsive to medical management, PMC should be considered particularly when anatomical conditions are favorable or in case of uncertainty regarding follow-up.

Termination of pregnancy should be considered in severe symptomatic stenotic lesions where PMC is not an option.

■ PERCUTANEOUS MITRAL COMMISSUROTOMY

- It is preferred to closed (transatrial) mitral commissurotomy (CMC) or open procedure with cardiopulmonary bypass (CPB). PMC is better than CMC in terms of valve area and long-term durability.
- It is contraindicated in the presence of a clot in LA.
- It is best performed after the 20th week of gestation to reduce fetal risk of radiation. The procedural time should be kept as short as possible and abdominal shield is used to reduce radiation risk. The patient should be warned of radiation risk to the fetus. Transesophageal echocardiographic (TOE or TEE) guidance may be useful to reduce radiation.

Closed Mitral Commissurotomy

It is still practiced in the developing world as it is less expensive and sophisticated cardiac catheterization facilities are not widely available.

Disadvantages:

- Uncontrolled procedure with risk of mitral regurgitation and even mitral valve rupture needing emergency CPB
- Cannot correct subvalvular deformity
- Risk of GA.

Cardiopulmonary bypass during pregnancy significantly affects mortality in the mother when compared to a nonpregnant state. However, it is associated with a high fetal mortality and late neurological impairment. Meticulous maintenance of blood pressure during bypass is important. Fetal well-being should be monitored continuously with a cardiotocograph.

When valvular surgery is required during pregnancy, cesarean section should be performed first if the fetus is viable.

Open Commissurotomy

When the valve can be conserved, it avoids the risks of prosthetic valves and also avoids the need for anticoagulation in patients in sinus rhythm.

Valve Replacement

Cardiopulmonary bypass during pregnancy is associated with a maternal mortality rate similar to that of the nonpregnant female (3–15%); however, there is significant morbidity including late neurological impairment in 3–6% of children, and fetal mortality remains high. For this reason, cardiac surgery is recommended only when medical therapy or interventional procedures fail and the mother's life is threatened. The best period for surgery is between the 13th and 28th weeks. During CPB, maintaining normothermia plays an important role in improving the fetal outcome as studies have shown that hypothermia (temperature $< 35^\circ\text{C}$) results in higher fetal mortality. With better care and outcome of prematurely born babies, caesarean delivery may be considered before CPB if the gestational age is > 26 weeks. As per practice advisory by the European Society of Cardiology, during CPB, the fetal heart rate and uterine tone should be monitored in addition to standard patient monitoring. Pump flow $> 2.5 \text{ L/min/m}^2$ and perfusion pressure $> 70 \text{ mm Hg}$ are mandatory to maintain adequate uteroplacental blood flow; pulsatile flow, although controversial, seems more effective for preserving uteroplacental blood flow. Maternal hematocrit $> 28\%$ is recommended to optimize the oxygen delivery. Normothermic perfusion, when feasible, is advocated, and state-of-the-art pH management is preferred to avoid

hypocapnia responsible for uteroplacental vasoconstriction and fetal hypoxia. CPB time should be minimized.

The choice of mechanical valve and a bioprosthesis is determined primarily by assessing the risk of anticoagulant-related bleeding with a mechanical valve versus the risk of structural valvular degeneration with a bioprosthesis. Bioprosthetic valves are less durable than mechanical valves but avoid the need for anticoagulation.

Antithrombotic Management

Oral anticoagulation is recommended for the following situations:

- Lifelong for all patients with mechanical valves
- Lifelong for patients with bioprostheses who have other indications for anticoagulation, e.g., AF
- For the first 3 months after insertion in all patients with bioprosthesis with a target INR of 2.5. aspirin (low dose: 75–100 mg) is also used as an alternative to anticoagulation for the first 3 months, but there are no randomized studies to support the safety of this strategy.

Delivery

Vaginal delivery is recommended whenever possible if the hemodynamic condition is stable at the end of pregnancy. Epidural analgesia helps in reducing sympathetic stimulation. However, titrated epidural boluses with close hemodynamic monitoring are needed to prevent precipitous drop in blood pressure. Hypotension is treated with cautious boluses of crystalloid solution and pure vasoconstrictors such as phenylephrine or noradrenaline.

The use of obstetric procedures (vacuum or outlet forceps) to shorten extraction time is recommended to reduce the total duration of labor, diminishing hemodynamic consequences. Hemodynamic monitoring is recommended in women with severe MS or LV dysfunction which is continued for 24 hours after delivery.

Cesarean Section

Advantage: Avoids the hemodynamic consequences of labor.

Disadvantages:

- Hemodynamic changes at induction of anesthesia due to vasodilatation and sympathetic stimulation
- Positive-pressure ventilation may worsen pulmonary hypertension and affects venous return
- Blood volume shift.

Close hemodynamic monitoring is needed particularly in severe stenosis as these patients are very sensitive to changes in preload and afterload and fluid and vasopressor therapy must be very carefully titrated against blood pressure. Advanced invasive monitoring including

continuous arterial BP monitoring and pulmonary artery catheterization maybe needed and should be ideally performed in specialized centers.

Spinal anesthesia is contraindicated in severe stenosis because of the uncontrolled hypotension. In less severe cases, spinal anesthesia with a single injection of 1 mL 0.25% bupivacaine and 10–20 µg of fentanyl or use of spinal catheter has been described. Routine antibiotic prophylaxis in patients with valvular heart disease undergoing uncomplicated vaginal delivery or cesarean section is not indicated. Prophylactic antibiotic therapy can be given at the beginning of labor and during delivery in patients at high risk, i.e., with previous endocarditis or when overt infection is present. Prophylactic antimicrobial therapy is indicated for invasive urinary tract or gastrointestinal procedures in pregnant women. Intravenous ampicillin and gentamicin, or oral amoxycillin can be used. In patients with history suggestive of penicillin allergy, one can use vancomycin and gentamicin.

Q. How will you maintain anticoagulation in a patient with a prosthetic valve?

Ans. The risk of embolization is greater with the prosthetic mitral valve than with the prosthetic aortic valve. Other risk factors for increased risk of embolization include AF, LV dysfunction, clotting disorder, and prior embolic events. With either type of prosthesis (bioprosthetic or mechanical) or valve location, the risk of emboli is higher in the first few months after valve insertion as the valve is not fully endothelialized. In most patients with a mechanical prosthesis, the target INR is 2.5–3.5. The target INR can be reduced to 2.0–3.0 in those patients with a new-generation AVR and no other risk factors for thromboembolic events.

The American College of Cardiology and American Heart Association (ACC/AHA) guidelines 2015 recommend the following:

- With mechanical aortic valve, vitamin K antagonist (VKA) therapy with a target INR of 2.5 if no risk factors (AF, prior thromboembolism, left ventricular dysfunction, or hypercoagulable state) are present (Class I recommendation; Level of evidence: B)
- VKA therapy with a target INR of 3.0 after:
 - Mechanical aortic valve replacement if risk factors are present (Class I recommendation; Level of evidence: B)
 - Mechanical aortic valve replacement with Starr-Edwards or disk valves other than Medtronic Hall if no risk factors are present (Class I recommendation; Level of evidence: B)
 - Mechanical mitral valve replacement with any type of valve (Class I recommendation; Level of evidence: B)

- Antiplatelet therapy: Aspirin at a dose of 75–100 mg/day in addition to warfarin in all patients with mechanical valves (Class I recommendation; Level of evidence: A)
- For patients with bioprosthetic heart valves: VKA therapy with a target INR of 2.5 (range 2.0–3.0):
 - For the first 3 months after bioprosthetic aortic valve replacement (Class IIb recommendation; Level of evidence B)
 - For the first 3 months after bioprosthetic mitral valve replacement (Class IIa recommendation; Level of evidence C)
 - *Antiplatelet therapy*: After bioprosthetic aortic or mitral valve replacement at a dose of 75–100 mg/day (Class IIa recommendation; Level of evidence: B).

For minor surgeries, where bleeding is easily controlled, anticoagulants should be adjusted to a target INR of 2.0 in the perioperative period.

For major surgical procedures, in which anticoagulant interruption is considered essential (INR < 1.5), patients should be admitted to hospital in advance and transferred to intravenous unfractionated heparin (UFH). Heparin is stopped 6 hours before surgery and resumed 6–12 hours after. Low molecular weight heparin (LMWH) can be given subcutaneously as an alternative preoperative preparation for surgery. Despite wide use and the positive results of observational studies, the safety of LMWHs has not been widely established and their efficacy has not been proved by controlled studies, particularly in patients at high risk of valve thrombosis. When LMWHs are used, they should be administered twice a day, using therapeutic rather than prophylactic doses, adapted to body weight and if possible according to monitoring of anti-Xa activity. LMWHs are contraindicated in case of renal failure.

Despite the low level of evidence for both strategies, the European Society of Cardiology (2007) favors the use of unfractionated intravenous heparin. Effective anticoagulation should be resumed as soon as possible.

In valve disease patients, all surgical procedures, even minor, require scrupulous asepsis and avoidance of wound hematoma formation. Antibiotic prophylaxis should be prescribed for those patients undergoing noncardiac procedures which put patients at high risk of bacteremia.

Q. How will you maintain anticoagulation during pregnancy?

Ans. Patients with MS with AF are at high risk for thromboembolism and vitamin K antagonists are the treatment of choice in nonpregnant patients. In patients with prosthetic valves, maternal mortality is estimated between 1 and 4% and is mostly related to thromboembolism. The risks are particularly high in patients with mitral valve prostheses. Therefore, these patients should be informed of the risks and they require careful risk assessment and very close monitoring of anticoagulant therapy if pregnancy occurs.

Choice of anticoagulants during pregnancy: In a large review, the risk of valve thrombosis during pregnancy was 3.9% with oral anticoagulants (OACs) throughout pregnancy, 9.2% when UFH was used in the first trimester and OACs in the second and third trimesters, and 33% with UFH throughout pregnancy. UFH throughout pregnancy is additionally associated with thrombocytopenia and osteoporosis. LMWHs are also associated with the risk of valve thrombosis.

Traditional teaching is that patients should be anticoagulated with heparin in the first trimester of pregnancy and then converted to warfarin for the remainder of the pregnancy. Warfarin should then be stopped prior to delivery. Because of different recommendations regarding anticoagulation during pregnancy, the decision is best taken after detailed discussion with the obstetrician, cardiologist, and patient. Vitamin K antagonists are titrated to an INR of 2–3 for best maternal protection. As vitamin K antagonists cross placenta, they increase the risk of miscarriage, prematurity, and embryopathy when used between the 6th and 12th weeks. However, there is some literature which supports use of warfarin < 5 mg in the first trimester. (The risk is lower when the warfarin dose is <5 mg/day; 2.6% vs. 8%.) Vitamin K antagonists are contraindicated during labor and delivery because of the risk of cerebral bleeding in the fetus.

Q. What are the implications of a prosthetic valve during labor?

Ans. Pregnancy in a woman with a mechanical valve is associated with an estimated maternal mortality of 1–4%, with death usually resulting from complications of prosthetic-valve thrombosis. A cardiology opinion is sought for 2D echocardiographic examination to assess valve function, clots, and LV function and to decide anticoagulation plan during pregnancy.

Vaginal delivery can be recommended:

- If the patient is not on oral anticoagulation at the onset of labor
- If there is no significant prosthetic dysfunction
- If there is no other significant cardiovascular disease, e.g., disease at another valve site, impairment of LV function, and aortic dilatation
- If a specialist obstetric anesthetist is available to provide epidural anesthesia.

Recommendations for the Evaluation and Care of Women of Childbearing Age with Mechanical Valve Prostheses who are taking Anticoagulants

Before conception:

- Clinical evaluation of cardiac functional status and previous cardiac events

- Echocardiographic assessment of ventricular and valvular function and pulmonary pressure
- Discussion of risks associated with pregnancy
- Discussion of risks and benefits associated with anticoagulant therapy
- Pregnancy planning.

The AHA/ACC 2013 Published Guidelines on Anticoagulation for Pregnant Patients with a Mechanical Prosthetic Heart Valve

Class I recommendations are:

- Therapeutic anticoagulation with frequent monitoring is recommended for all pregnant patients with a mechanical prosthesis (Level of Evidence: B).
- Warfarin is recommended in pregnant patients with a mechanical prosthesis to achieve therapeutic INR in the second and third trimesters (Level of Evidence: B).
- Discontinuation of warfarin with initiation of intravenous UFH (with an APTT > 2 control) is recommended before planned vaginal delivery in pregnant patients with a mechanical prosthesis (Level of Evidence: C).
- Low-dose aspirin (75–100 mg) once per day is recommended for pregnant patients in the second and third trimesters with either a mechanical prosthesis or a bioprosthesis (Level of Evidence: C).

Class IIa recommendations:

- Continuation of warfarin during the first trimester is reasonable for pregnant patients with a mechanical prosthesis if the dose of warfarin to achieve a therapeutic INR is <5 mg/day, after full discussion with the patient about risks and benefits (Level of Evidence: B).
- Dose-adjusted LMWH at least twice per day (with a target anti-Xa level of 0.8–2U/mL, 4–6 hours post dose) during the first trimester is reasonable for pregnant patients with a mechanical prosthesis if the dose of warfarin is >5 mg/day to achieve a therapeutic INR (Level of Evidence: B).
- Dose-adjusted continuous intravenous UFH (with an APTT at least 2 times control) during the first trimester is reasonable for pregnant patients with a mechanical prosthesis if the dose of warfarin is >5 mg/day to achieve a therapeutic INR (Level of Evidence: B).

Delivery

- Heparin should be discontinued at the start of labor.
- Restart heparin therapy 4–6 hours after delivery if no contraindications.
- Oral anticoagulation should be resumed after 24 hours if no concerns regarding bleeding with IV UFH continued

till desired INR is reached (>2) when IV UFH can be stopped.

- If labor occurs preterm while the patient is still on oral anticoagulants, a cesarean section should be performed after reducing the INR to 2.0. A vaginal delivery should be avoided under oral anticoagulation because of the danger of fetal intracranial bleeding.

Prosthetic Thrombosis

It is a dreaded complication with high mortality and should be suspected in any patient who has a prosthetic valve developing worsening of cardiac condition.

Diagnosis: Transthoracic echocardiography and/or transesophageal echo.

Management: Options are surgery or fibrinolysis. Surgery is high risk because it is most often performed in emergency conditions and is reintervention. Fibrinolysis carries risks of bleeding, systemic embolism, and recurrent thrombosis.

Q. What are the implications of CPB in pregnancy?

Ans. The risk to mother is the same as in a nonpregnant state. However, it confers high risk to the fetus.

Fetal risk is due to:

- Increased uterine contraction
- Placental hypoperfusion.

The risk of teratogenesis is highest in the first trimester due to drugs and possibly CPB, and surgery during this period is avoided. High flow, high pressure normothermic bypass, tocolytics, and fetal monitoring are advocated. The fetus has bradycardic response to CPB and is thought to be due to hypoperfusion. Tocolytics with β -agonist action are avoided. Magnesium is preferred.

Q. What are the implications of various cardiovascular diseases in pregnancy and what are various risk stratification scores?

Ans. A pregnant lady can present with many congenital and acquired cardiovascular diseases of which valvular heart disease is the most common. Physiologic changes in pregnancy, namely decreased peripheral vascular resistance, increased plasma volume, and increased CO, increase flow across valve and thus increase transvalvular gradient across pre-existing valvular lesions. These changes can get further exacerbated during labor. Hence, counseling and risk stratification are a must for these patients. Presently, there are three widely used risk stratification scores to quantify maternal cardiac risk: (1) Cardiac Disease in Pregnancy (CARPREG) risk score, (2) Zwangerschap bij Aangeboren HARTafwijkingen I (ZAHARA) risk score, and World Health Organization (WHO) classification.

A prospective study comparing the above three scores in pregnant patients with congenital heart disease showed better performance of the WHO score.

CARPREG score consists of four risk factors, each carrying one point. The more the risk factors, the more the risk of maternal cardiac complications. The risk factors are as follows:

1. Prior cardiac event like transient ischemic attack (TIA), heart failure, or arrhythmias
2. NYHA classes III and IV or presence of cyanosis
3. Left ventricular outflow obstruction
4. Left ventricular EF < 40%.

In 2018, Silverside and colleagues revised CARPREG score to fine-tune the assessment of the risk of cardiac complications in pregnancy. CARPREG II risk score is based on 10 predictors with assigned weighted score. The risk factors are as follows:

1. Prior cardiac event such as TIA, heart failure, or arrhythmias—3 points
2. NYHA classes III and IV or presence of cyanosis—3 points
3. Mechanical valve—3 points
4. Ventricular dysfunction—2 points

5. High-risk left-sided valve disease/left ventricular outflow obstruction—2 points
6. Pulmonary hypertension—2 points
7. Coronary artery disease—2 points
8. High-risk aortopathy—2 points
9. No prior cardiac intervention—1 point
10. Late pregnancy assessment—1 point.

ZAHARA is a more comprehensive weighted risk score but relates to congenital heart disease. Risk score has eight risk factors with different weightage:

1. History of arrhythmia—1.5 points
2. Cardiac medication prior to pregnancy—1.5 points
3. NYHA class III/IV—0.75 point
4. Left ventricular outflow obstruction—2.5 points
5. Systemic atrioventricular valve regurgitation (moderate or severe)—0.75 point
6. Pulmonic atrioventricular valve regurgitation (moderate or severe)—0.75 point
7. Presence of mechanical prosthetic valve—4.25 points
8. Cyanotic heart disease (corrected or uncorrected)—1 point.

The modified WHO classification of maternal cardiovascular risk is more elaborate (Table 3).

TABLE 3 : Modified WHO classification of maternal cardiovascular risk.

Risk stratification	Cardiac lesions
<i>Class I:</i> No detectable increased risk of maternal mortality and no/minimal increase in maternal morbidity	• Uncomplicated, small or mild pulmonary stenosis; ventricular septal defect; patent ductus arteriosus; mitral valve prolapse with no more than trivial mitral regurgitation • Successfully repaired simple lesions (atrial or ventricular septal defect, patent ductus arteriosus, anomalous pulmonary venous drainage) • Isolated ventricular extrasystoles and atrial ectopic beats
<i>Class II</i> (if otherwise well and uncomplicated): Small increased risk of maternal mortality or moderate increase in morbidity	• Unoperated atrial or ventricular septal defect • Repaired tetralogy of Fallot • Most arrhythmias
<i>Classes II to III:</i> Depending on patient	• Mild ventricular impairment • Heart transplantation • Hypertrophic cardiomyopathy • Native or tissue valvular heart disease not considered WHO I or IV • Repaired coarctation • Marfan syndrome without aortic dilatation • Bicuspid valve with aorta <45 mm
<i>Class III:</i> Significantly increased risk of maternal mortality or severe morbidity Need expert cardiac and obstetric pre-pregnancy, antenatal, and postnatal care	• Mechanical valve • Systemic right ventricle • Fontan circulation • Unrepaired cyanotic heart disease • Other complex CHD • Marfan syndrome with aorta 40–45 mm • Bicuspid aortic valve with aorta 45–50 mm
<i>Class IV:</i> Pregnancy is contraindicated	• Pulmonary hypertension/Eisenmenger syndrome, ventricular ejection fraction <30% or systemic ventricular dysfunction with NYHA classes III and IV • Severe MS, severe symptomatic AS • Marfan syndrome with aorta >45 mm • Bicuspid aortic valve with aorta >50 mm • Native severe coarctation • Prior peripartum cardiomyopathy with any residual impairment of ventricular function

SUGGESTED READING

1. Balci A, Sollie-Szarynska KM, van der Bijl AG, Ruys TP, Mulder BJ, Roos-Hesselink JW, et al. Prospective validation and assessment of cardiovascular and offspring risk models for pregnant women with congenital heart disease. *Heart*. 2014;100(17):1373-81.
2. Baumgartner H, Hung J, Bermejo J, Chambers JB, Evangelista A, Griffin BP, et al. Echocardiographic assessment of valve stenosis: EAE/ASE recommendations for clinical practice. *Eur J Echocard*. 2009;10:1-25.
3. Bonow RO, Carabello BA, Chatterjee K, de Leon AC Jr, Faxon DP, Freed MD, et al. Writing Committee Members; American College of Cardiology/American Heart Association Task Force, 2006. 2008 Focused update incorporated into the ACC/AHA 2006 guidelines for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2008;118(15):e523-661.
4. Butchart EG, Gohlke-Bärwolf C, Antunes MJ, Tornos P, De Caterina R, Cormier B, et al. Working Groups on Valvular Heart Disease, Thrombosis, and Cardiac Rehabilitation and Exercise Physiology, European Society of Cardiology. Recommendations for the management of patients after heart valve surgery. *Eur Heart J*. 2005;26:2463-71.
5. Carabello BA. Modern management of mitral stenosis. *Circulation*. 2005;112:432-7.
6. De Caterina R, Husted S, Wallentin L, Agnelli G, Bachmann F, Baigent C, et al. Anticoagulants in heart disease: current status and perspectives. *Eur Heart J*. 2007;28:880-913.
7. Elkayam U, Bitar F. Valvular heart disease and pregnancy part I: native valves. *J Am Coll Cardiol*. 2005;46:223-30.
8. Elkayam U, Bitar F. Valvular heart disease and pregnancy part II: prosthetic valves. *J Am Coll Cardiol*. 2005;46: 403-10.
9. ESC Guidelines on the management of cardiovascular diseases during pregnancy. The Task Force on the Management of Cardiovascular Diseases during Pregnancy of the European Society of Cardiology (ESC). *Eur Heart J* 2011;32:3147-97.
10. Guidelines for the diagnosis of rheumatic fever. Jones Criteria, 1992 update. Special Writing Group of the Committee on Rheumatic Fever, Endocarditis, and Kawasaki Disease of the Council on Cardiovascular Disease in the Young of the American Heart Association. *JAMA*. 1992;268:2069-73.
11. Joubert IA, Dyer RA. Anaesthesia for the pregnant patient with acquired valvular heart disease. *Update Anaesthesia*. 2005;19(9).
12. Mulder BJ, Bleker OP. Valvular heart disease in pregnancy. *N Engl J Med*. 2003;349:1387.
13. Nishimura RA, Carabello BA, Faxon DP, Freed MD, Lytle BW, O'Gara PT. ACC/AHA 2008 Guideline update on valvular heart disease: focused update on infective endocarditis: *J Am Coll Cardiol*. 2008;52(8):676-85.
14. Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP 3rd, Guyton R, et al. 2014 AHA/ACC guideline for the management of patients with valvular heart disease: executive summary: a report of the American College of Cardiology/American heart association task force on practice guidelines. *Circulation*. 2014;129(23):2440-92.
15. Regitz-Zagrosek V, Roos-Hesselink JW, Bauersachs J, Blomstrom-Lundqvist C, Cifkova R, De Bonis M, et al. 2018 ESC Guidelines for the management of cardiovascular diseases during pregnancy. *Eur Heart J*. 2018;39:3165-241.
16. Vahanian A, Palacios IF. Review: clinical cardiology. New frontiers. Percutaneous approaches to valvular disease. *Circulation*. 2004;109(13):1572-9.

Ischemic Heart Disease

Jigeeshu Vasistha Divatia, Jeson Rajan Doctor, Aparna Chatterjee

A 70-year-old hypertensive male patient with history of chest pain is posted for a total gastrectomy.

Q. What are the known risk factors for the development of ischemic heart disease (IHD)?

Ans. Age, male gender, and a positive family history are risk factors that cannot be modified. Hypercholesterolemia, hypertension, and cigarette smoking are risk factors for which interventions have been proven to lower IHD risk. Sedentary lifestyle, diabetes mellitus, and obesity are the risk factors for which interventions are likely to lower IHD risk.

Q. Describe the normal coronary blood flow (CBF).

Ans. The resting CBF averages about 225 mL/min, which is 4–5% of the total cardiac output in normal adults. The CBF increases three- to fourfold to supply the extra nutrients needed by the heart at maximum exercise level. The CBF is determined by the pressure gradient between the aorta and the ventricles. There are phasic changes in CBF during systole and diastole in the left ventricle. The blood flow falls to a low value during systole, especially in the subendocardial area, because of compression of left ventricular muscle around the intramuscular vessels and the small pressure gradient between the aorta and the left ventricle (aortic systolic pressure minus the left ventricular systolic pressure). During diastole, the cardiac muscle relaxes and no longer obstructs the blood flow through the left ventricular capillaries. The pressure gradient (coronary perfusion pressure) is high during diastole [aortic diastolic pressure minus the left ventricular end-diastolic pressure (LVEDP)]. Thus, CBF occurs maximally in diastole. Conditions and interventions that prolong the diastolic time are beneficial, while those that reduce the diastolic filling time are detrimental. The phasic changes of the CBF are much less in the right ventricle because the force of contraction of the right ventricle is much weaker than that of the left ventricle and the pressure gradient is

high between the aorta and the right ventricle during the entire cardiac cycle.

Q. Describe the coronary anatomy.

Ans. The right coronary artery system is dominant in 80–90% of people and supplies the sinoatrial node, atrioventricular (AV) node, and the right ventricle. Right-sided coronary artery disease (CAD) often manifests as heart block and dysrhythmias (**Fig. 1**).

The left main coronary artery gives rise to the circumflex artery and left anterior descending artery, which supply the majority of the interventricular septum and the left ventricular wall. Significant stenosis of the left main coronary artery (left main disease), the proximal circumflex, and left anterior descending arteries (left main equivalent) may severely depress myocardial function.

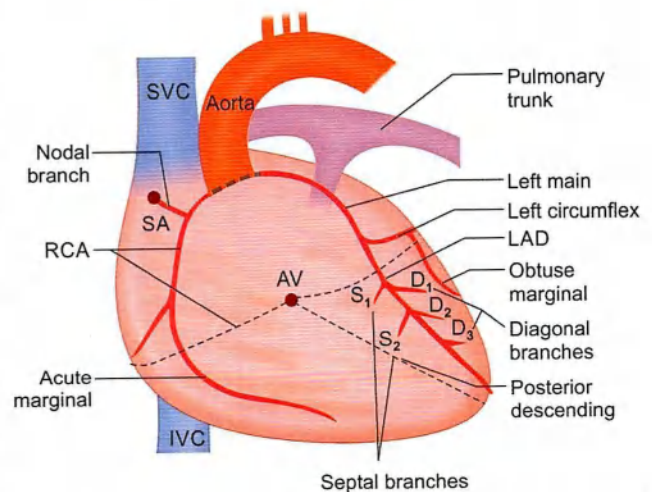


Fig. 1: Coronary artery system (AV: atrioventricular; IVC: inferior vena cava; LAD: left anterior descending; RCA: right coronary artery; SA: sinoatrial; SVC: superior vena cava).

Q. Explain the determinants of myocardial oxygen demand and delivery.

Ans. Myocardial oxygen demand is determined by wall tension and contractility. Wall tension (T) is the product of intraventricular pressure (P), radius (r), and the wall thickness (h): ($T = P \times r/2h$) (Laplace's law). Increased ventricular end-diastolic volume (preload) and increased blood pressure (BP) (afterload) increase the wall tension and O₂ demand. Increase in contractility in response to sympathetic stimulation or inotropic medications also increases O₂ demand.

Increase in the heart rate increases the myocardial contractility, but decreases the ventricular diameter and the wall tension. Thus, the increase in oxygen demand caused by enhanced contractility is in large part offset by the reduction in oxygen demand that accompanies the reduced wall tension. However, the oxygen demand increases because of more contractions performed per minute.

Myocardial oxygen delivery is determined by oxygen content and CBF. Oxygen content can be calculated by the following equation:

$$\text{O}_2 \text{ content} = [1.34 \text{ mL O}_2/\text{g of hemoglobin} \times \text{hemoglobin (g/dL)} \times \text{saturation}] + (0.003 \times \text{PaO}_2)$$

The oxygen content is decreased in anemia and hypoxemia.

Coronary blood flow is determined by coronary perfusion pressure, the time available for perfusion, and the patency of coronary arteries. Coronary perfusion pressure = Aortic diastolic pressure – LVEDP. Coronary perfusion pressure is thus reduced by diastolic hypotension, left ventricular hypertrophy (LVH), and increased LVEDP. The diastolic time for perfusion is decreased with tachycardia. The patency of the coronary arteries can be influenced by vasospasm or coronary occlusion caused by atherosclerosis.

Q. What is the pathophysiology of myocardial ischemia?

Ans. Myocardial ischemia occurs when CBF is inadequate to meet the needs of the myocardium. The main coronary artery epicardial branches have lumens that are 2–4 mm in diameter. In the absence of collaterals, exertional angina occurs when the lumen area is reduced to 1 mm (50–60% reduction in diameter or 75% reduction in the cross-sectional area) and angina at rest occurs when the luminal area is reduced to 0.65 mm (75% reduction in diameter or 90% reduction in cross-sectional area). Most of the sclerotic lesions are eccentrically located so the remainder of the arterial wall is responsive to vasoactive stimuli and is capable of contraction. Therefore, the severity of the stenosis is dynamic and influenced by the vasomotor activity of the free arterial wall.

Nonstenotic causes of myocardial ischemia include aortic valve disease, LVH, ostial occlusion, coronary embolism, coronary arteritis, and vasospasm.

Q. What are the clinical manifestations of myocardial ischemia?

Ans. The clinical manifestations of myocardial ischemia are varied. Angina pectoris with or without the signs of arrhythmias or heart failure is assumed to be the classic manifestation of myocardial ischemia. However, myocardial ischemia may present as ventricular failure or arrhythmias without angina or may remain clinically silent. The dynamic nature of coronary stenosis accounts for the changes in the caliber of a stenosis that may produce rest pain at one time and angina with varying degrees of exercise at other times.

Q. How can the angina be graded?

Ans. The Canadian Cardiovascular Society introduced a grading system for angina:

- *Class I:* Angina with strenuous or rapid prolonged exertion at work or recreation.
- *Class II:* Angina with walking or climbing stairs rapidly, walking uphill or walking more than two blocks on the level and climbing more than one flight of ordinary stairs at a normal pace.
- *Class III:* Angina with walking one to two blocks on the level and climbing one flight of stairs at a normal pace.
- *Class IV:* Angina may be present at a very low level of physical activity or at rest.

Q. What is unstable angina? Describe the pathogenesis of a perioperative myocardial infarction (PMI)?

Ans. Unstable angina includes a spectrum of syndromes between stable angina and myocardial infarction (MI). The angina attacks may increase in frequency or severity, persist longer, respond less to nitrates, and may occur at rest or on minimal exertion.

An MI is usually caused by plaque disruption, platelet aggregation, vasoconstriction, and thrombus formation at the site of an atheromatous plaque in a coronary artery. Theoretically, sudden increases in myocardial O₂ demand (e.g., tachycardia and hypertension) or decreases in O₂ supply (e.g., hypotension, hypoxemia, anemia, tachycardia, coronary occlusion) can precipitate MI in patients with IHD. Most perioperative ischemic events are unrelated to hemodynamic perturbations, suggesting that intracoronary events may be important in the genesis of perioperative ischemia. Complications of MI include dysrhythmias, hypotension, congestive heart failure (CHF), acute mitral regurgitation, pericarditis, ventricular thrombus formation, ventricular rupture, and death.

Q. What clinical factors increase the risk of a perioperative major adverse cardiac events (MACE), i.e., death or MI following noncardiac surgery?

Ans. There are major, intermediate, and minor clinical predictors based on the algorithm for risk stratification and appropriate use of diagnostic testing of the American College of Cardiology/American Heart Association (ACC/AHA) Task Force on Perioperative Evaluation of Cardiac Patients Undergoing Noncardiac Surgery.¹ The guidelines integrate clinical risk factors, exercise capacity, and the surgical procedure in the decision process.

Major factors (markers of unstable CAD):

- Acute MI (<7 days) or recent MI (7–30 days)
- Unstable or severe angina class III and IV
- Decompensated heart failure [New York Heart Association (NYHA) functional class IV worsening or new onset heart failure].
- *Significant arrhythmias:*
 - High-grade AV block
 - Mobitz type II AV block
 - Third-degree AV block
 - Symptomatic ventricular arrhythmias
 - Supraventricular arrhythmias (including atrial fibrillation) with uncontrolled ventricular rate (heart rate >100 beats per minute at rest)
 - Symptomatic bradycardia
 - Newly recognized ventricular tachycardia.

Intermediate factors:

- History of IHD (excluding revascularization)
- History of congestive cardiac failure (CCF)
- History of a stroke or transient ischemic attack (TIA)
- Preoperative insulin therapy (diabetes)
- Serum creatinine >2 mg% (renal failure).

Minor factors (increased probability of CAD):

- Familial history of CAD
- Polyvascular status
- Uncontrolled systemic hypertension
- Hypercholesterolemia
- Smoking
- Electrocardiogram (ECG) abnormalities (arrhythmia, LVH, and bundle branch block)
- Postinfarction (>3 months), asymptomatic without treatment
- Post-coronary artery bypass grafting (post-CABG) or percutaneous transluminal coronary angioplasty (PTCA) >3 months and <6 years, and no symptoms of angina and not on antianginal therapy.

Terminologies/definition:

- *Emergency procedure:*
 - Life or limb is threatened without surgery

- No time, or about <6 hours, minimal clinical evaluation

- *Urgent procedure:* About 6–24 hours' time for a limited clinical evaluation.
- *Time-sensitive procedure:*
 - *Delay of no >1–6 weeks:*
 - Most oncologic procedures would fall into this category
- *An elective procedure:* Procedure could be delayed for up to 1 year.
- *A low-risk procedure:* Combined surgical and patient characteristics predict a risk of a MACE (death or MI) of <1%.

Q. How does the type of surgery influence the risk stratification for perioperative ischemia?

Ans.

- High-risk procedures (risk of perioperative MACE >5%) include emergent major operations, aortic and major vascular procedures, peripheral vascular surgeries, and anticipated prolonged procedures associated with large fluid shifts and/or blood loss.
- Intermediate-risk procedures (risk of MACE 1–5%) include carotid endarterectomy, head and neck surgery, intraperitoneal and intrathoracic surgery, orthopedic surgery, and prostate surgery.
- Low-risk procedures (risk of perioperative MACE <1%) include endoscopic procedures, superficial procedures, cataract surgery, and breast surgery and ambulatory surgery.

Q. How can cardiac function be evaluated on history and physical examination?

Ans. If a patient's exercise capacity is excellent, even in the presence of IHD, the chances are good that the patient will be able to tolerate the stresses of surgery. Poor exercise tolerance in the absence of pulmonary or other systemic disease indicates an inadequate cardiac reserve. All patients should be questioned about their ability to perform daily activities, such as is described in the Canadian classification of angina pectoris or the NYHA classification of exercise tolerance.

Assessment of functional capacity: The Duke Activity Status Index (DASI) and approximate metabolic equivalents (METs—1 MET represents an oxygen consumption of 3.5 mL/kg/min)

- *1–4 METs:*
 - Standard light home activities
 - Walk around the house
 - Take care of yourself—eating, dressing, bathing, and using the toilet

- 5–9 METs:
 - Climb a flight of stairs and walk up a hill
 - Walk one or two blocks on level ground
 - Run a short distance
 - Moderate activities (golf, dancing, and mountain walk)
 - Have sexual relations
- >10 METs:
 - Strenuous sports (swimming, tennis, and bicycle)
 - Heavy professional/domestic work such as scrubbing floors, lifting, or moving heavy furniture
 - Signs and symptoms of CHF, including dyspnea, orthopnea, paroxysmal nocturnal dyspnea, peripheral edema, jugular venous distention, a third heart sound, rales, and hepatomegaly must be recognized preoperatively.

Q. What is the definition of recent and prior MI?

Ans. Recent monitoring and therapeutic strategies including aggressive recanalization as well as preoperative optimization of patients for surgery significantly decrease the risk for reinfarction even in patients with recent MI. Based on the decreased risk for perioperative reinfarction, the American College of Cardiology National Database defines an acute MI as one that is ≤ 7 days old. Recent MI is defined as MI occurring within >7 days but <30 days. Prior MI is defined as an MI that occurred >30 days before the surgery.

Q. What ECG findings support the diagnosis of IHD?

Ans. The resting 12-lead ECG remains a low cost-effective screening tool in the detection of IHD. It should be evaluated for the presence of ST segment depression or elevation, T wave inversion, old MI as demonstrated by Q waves, disturbances in conduction and rhythm, and LVH. Ischemic changes in leads II, III, and avF suggest right CAD, leads I and avL monitor the circumflex artery distribution and leads V_3 to V_5 look at the distribution of the left anterior descending artery.

Q. What is the significance of the presence of IHD?

Ans. Patients with unstable or severe stable angina (Class III to IV) or recent MI without adequate revascularization are at a high risk for developing PMI and prior to undergoing elective noncardiac surgery should be referred for medical evaluation, coronary angiography, and revascularization.

Patients with stable angina (Class I to II) or prior MI need further risk stratification and intervention based on their exercise capacity and the severity of the surgery. Patients with poor exercise capacity (<4 METs) or patients with good exercise capacity (>4 METs), but who are to undergo a high-risk surgery, need noninvasive testing to evaluate reversible ischemic myocardial areas during exercise.

Coronary angiography and appropriate revascularization should be done before noncardiac surgery in patients with exercise-induced myocardial ischemia.

Q. What tests can help further evaluate patients with known or suspected IHD?

Ans. Further tests to evaluate a patient with known or suspected IHD/CAD include:

- Resting ECG
- Stress ECG/stress test
- Resting echocardiogram
- Stress echocardiogram/dobutamine stress echocardiogram
- Cardiopulmonary exercise testing (CPET)
- Dipyridamole-thallium imaging
- Coronary angiography.

Resting Electrocardiogram

According to the AHA guidelines,¹ a resting 12-lead ECG:

- Is reasonable for patients with known CAD or other structural heart disease, except in low-risk surgery?
- May be considered for asymptomatic patients, undergoing moderate- to high-risk surgery.
- Is not useful for asymptomatic patients undergoing low-risk surgical procedures?

Exercise ECG or Stress Test

Of the noninvasive cardiac tests of the wide variety of tests advocated, the exercise ECG or stress test is the most cost-effective means of detecting CAD (sensitivity $\approx 70\%$, specificity $\approx 70\%$). It simulates sympathetic nervous system stimulation that may accompany perioperative events such as laryngoscopy and surgical stimulation.

An exercise ECG:

- Provides an objective estimate of functional capacity
- Can detect myocardial ischemia or arrhythmia
- Can be used to estimate perioperative cardiac risk and long-term prognosis.

Interpretation of the exercise ECG is based on: (1) the duration of exercise that the patient is able to perform, (2) the maximum heart rate that is achieved (target heart rate achieved), (3) the time of onset of ST segment depression on the ECG, (4) the degree of ST segment depression, and (5) the time until resolution of the ST segment depression during the recovery period.

A normal exercise ECG indicates that the coronary circulation is reasonably adequate. The usefulness of this technique is, however, limited by the fact that high-risk patients may be unable to exercise to a level required to achieve target rates, and exercise may be contraindicated in others. Also, approximately 10% of the adult population with normal coronary arteries may develop ST segment changes on the exercise ECG that resemble changes observed in the

patients with IHD. For this reason, use of the exercise ECG in an asymptomatic patient is of doubtful value.

As per the AHA guidelines:

- A stress test is not necessary in patients with an elevated risk but moderate-to-excellent functional capacity (>4 METs)
- In patients with <4 METs or unknown functional capacity, it is reasonable to perform a stress test or a cardiac imaging to assess for myocardial ischemia
- In low-risk noncardiac surgeries, routine stress testing is not useful.

Echocardiography

It is used to evaluate left ventricular function (LVF) and measure the ejection fraction.

As per the AHA guidelines:¹

- Routine preoperative evaluation of LVF is not recommended
- In patients with dyspnea of unknown origin or heart failure with worsening dyspnea or other change in clinical status, it is reasonable to evaluate LVF
- Reassessment of LVF in clinically stable patients may be considered.

Pharmacological Stress Testing

It reproduces the cardiovascular effects of exercise and is particularly useful in patients who are unable to exercise:

- **Dobutamine stress echocardiography:** Stress echocardiography has the advantage of being able to assess both regional wall motion abnormalities resulting from induced myocardial ischemia and LVF (in situations of increased myocardial oxygen demand). It offers the best prediction for perioperative events, with a negative predictive value close to 100% (negative or normal stress ECHO rules out CAD) and a positive predictive value up to 38% among intermediate or high-risk patients. Areas of wall motion abnormality are considered at risk for ischemia.
- In dipyridamole thallium myocardial imaging, dipyridamole (an adenosine agonist that increases blood flow in normal coronary arteries) is used to simulate the effects of exercise. Images are acquired by the coadministration of a radioisotope, such as thallium, which gets distributed to the myocardium in direct proportion to CBF. A reversible defect (i.e., a defect seen on the initial stress image that disappears with rest) indicates the presence of CAD (sensitivity 85–90% and specificity 85–90%).

According to the AHA guidelines:

- Routine screening with noninvasive stress testing is not useful in low-risk noncardiac surgery.
- Reasonable to perform these tests in a patient with poor functional capacity and an elevated risk if it will change the management.

Cardiopulmonary Exercise Testing

Cardiopulmonary exercise testing carried out in high-risk surgeries such as abdominal aortic aneurysm surgery, major abdominal surgery (including abdominal aortic aneurysm resection), hepatobiliary surgery, complex hepatic resection, lung resection, colorectal, bladder, and renal cancer surgery. A low anaerobic threshold was found to be predictive of perioperative cardiovascular complications. An anaerobic threshold of approximately 10 mL O₂/kg/min was proposed as the optimal discrimination point, with a range in these studies of 9.9–11 mL O₂/kg/min.

The AHA guidelines currently recommend the use of CPET in patients undergoing elevated risk procedures.

Coronary Angiography

Coronary angiography is an invasive procedure, and is indicated only in cases of unstable coronary syndromes, or uncertain stress tests in high-risk patients undergoing major surgery, or when there is a possible indication for coronary revascularization. Cardiac catheterization on its own has been reported to carry a mortality rate of 0.01–0.5% and of serious morbidity ranging from 0.03 to 0.25%.²

As per the AHA guidelines, routine preoperative coronary angiography is not recommended.

Q. How would you stratify risk of anesthesia and surgery for this patient? What are the risk factors in the Lee revised cardiac risk index (RCRI)?

Ans. For quantification of risk, three key questions need to be considered:

1. Are there modifiable operative risk factors?
2. If the patient is at high-risk, should the elective operation be modified, delayed, or cancelled?
3. Is coronary revascularization and/or valve surgery indicated?

An overall assessment of perioperative cardiac risk requires consideration of the type of surgery planned, the presence and type of specific clinical indicators of CAD, and the patient's functional status.

Historically, the Goldman multifactorial cardiac risk index was used to stratify patients according to cardiac risk. The modification of the Goldman index, proposed by Detsky et al., provides improved risk assessment in patients undergoing vascular surgery. In 1999, Lee and colleagues described RCRI and is reported to be superior to both previous indices (**Table 1**).

TABLE 1: Risk factors and mortality.

No. of risk factors	Event rate
0	0.4%
1	0.9%
2	6.6%
>3	11%

Six risk factors were identified in Lee's RCRI.³ This index is now incorporated into the AHA/ACC guidelines for preoperative assessment:

1. High-risk surgical procedure, such as intrathoracic, intraperitoneal, or suprainguinal vascular reconstruction
2. History of IHD (excluding revascularization)
3. History of CCF
4. History of a stroke or TIA
5. Preoperative insulin therapy (diabetes)
6. Serum creatinine >2 mg% (renal failure).

The presence of each risk factor contributed one point to the index. The risk of cardiac complications depended on the number of risk factors (**see Table 1**).

Gupta perioperative cardiac risk calculator: It is developed and validated by Gupta et al.⁴ for the prediction of cardiac risk after noncardiac surgery. They used the American College of Surgeons National Surgical Quality Improvement Program (NSQIP) Database involving 211,410 patients from 180 hospitals from the 2007 data set and validated it with the 2008 data set, which included 257,385 patients. The five predictors of PMI or cardiac arrest identified included the type of surgery, dependent functional status, abnormal creatinine, American Society of Anesthesiologists' class, and increasing age. It has a higher accuracy in comparison with the RCRI developed in 1999, and is available as an interactive risk calculator to enable calculating the risk at the bedside or clinics with simplicity and accuracy. This is readily available as an online calculator to assess and predict cardiovascular risk.

Other risk indices are not commonly used, but are enumerated in **Table 2**.

TABLE 2: Various cardiac risk indices.

	Goldman 1977	Detsky 1986	Eagle 1989	Lee 1999
Age >70 years	5	5	1	
Emergency surgery	4	10	–	
Intraperitoneal, intrathoracic, or aortic surgery	3	–	–	
Canadian Cardiovascular Society Class III angina	–	10	–	
Canadian Cardiovascular Society Class IV angina	–	20	–	
Unstable angina in previous 6 months	–	10	–	
Any angina	–	–	1	
Myocardial infarction in previous 6 months	10	10	–	

Contd...

	Goldman 1977	Detsky 1986	Eagle 1989	Lee 1999
Myocardial infarction >6 months ago	–	5	–	
Q-wave on ECG	–	–	1	
Third heart sound or jugular vein distension	11	–	–	
Pulmonary edema in previous 7 days	–	10	–	
Any history of pulmonary edema	–	5	–	
Significant aortic stenosis (i.e., gradient >50 mm Hg)	3	20	–	
ECG rhythm other than sinus with or without APBs	7	5	–	
>5 PVBs/min documented on ECG before surgery	7	5	–	
History of ventricular ectopy	–	–	1	
Diabetes mellitus	–	–	1	
Poor general medical status	3	5	–	
High-risk surgery				1
History of ischemic heart disease				1
History of congestive cardiac failure				1
History of cerebrovascular disease				1
Preoperative treatment with insulin				1
Preoperative serum creatinine >2 mg%				1
Total possible points	53	120	5	6
Risk class I	0–5 (1%)	0–15 (0.43)	0 (0–3%)	0 (0.4–0.5%)
Risk class II	6–12 (6.6%)	20–30 (3.38)	1–2 (6–16%)	1 (0.9–1.3%)
Risk class III	13–25 (13.8%)	> 30 (10.60)	>3 (25–50%)	2 (4–7%)
Risk class IV	>26 (78%)	–	–	>2 (9–11%)

(APB: atrial premature beat; ECG: electrocardiogram; PVB: premature ventricular beat).

Contd...

Q. What are the recommendations for perioperative cardiac risk assessment of patients undergoing noncardiac surgery?

Ans. The recommendations⁵ for patients depending on the nature of surgery are as follows:

- *Emergency surgery:* Recommend against delaying surgery for a preoperative cardiac risk assessment.
- *Urgent or semi-urgent surgery:* Recommend preoperative cardiac risk assessment only if patients' history or physical examination suggests:
 - Potential undiagnosed severe obstructive intracardiac abnormality
 - Severe pulmonary hypertension
 - An unstable cardiovascular condition
- *Elective noncardiac surgery:* Recommend preoperative cardiac risk assessment in patients:
 - Who are 45 years of age or older
 - Who are 18–44 years of age with known significant cardiovascular disease.

Recommend communicating to patients their perioperative cardiac risk.

Methods of Preoperative Cardiac Risk Assessment⁵

- *Revised cardiac risk index:* Recommend using this index over other indices for risk assessment. An online Gupta risk calculator is a web-based application available online for calculation of perioperative cardiac risk.
- *Self-reported functional capacity:* Limited data to guide perioperative cardiac risk assessment based on self-reported functional capacity to determine their METs.
- *Cardiac biomarkers:* Recommend measuring NT-proBNP (N-terminal fragment pro Brain Natriuretic peptides) or BNP (Brain Natriuretic peptide) before noncardiac surgery in patients who:
 - Are 65 years of age or older
 - Are 45–64 years of age with significant cardiovascular disease
 - Have an RCRI score of ≥ 1

These are available as point-of-care testing. The recommended higher normal values for NT-proBNP is 300 mg/L and BNP is 92 mg/L.
- *Resting echocardiography:* Recommend against performing preoperative resting echocardiography.
- *Coronary computed tomographic angiography (CCTA):* Recommend against performing preoperative CCTA.
- *Exercise stress testing and CPET:* Recommend against performing preoperative exercise stress testing or preoperative CPET.

- *Pharmacological stress echocardiography and radionuclide imaging:*

- Recommend against performing preoperative pharmacological stress echocardiography
- Recommend against performing preoperative pharmacological stress radionuclide imaging.

Q. Discuss the perioperative interventions for perioperative risk modification.

Ans. Initiation of therapy: The Canadian Cardiovascular Society⁵ has recommended against the initiation of acetylsalicylic acid (ASA), β -blockers [based on results of POISE (Perioperative Ischemia Study Evaluation) trial increasing the incidence of stroke due to hypotension], alpha-2 agonists, and calcium channel blockers in the perioperative period (when the patient is not on chronic therapy with any of these medications).

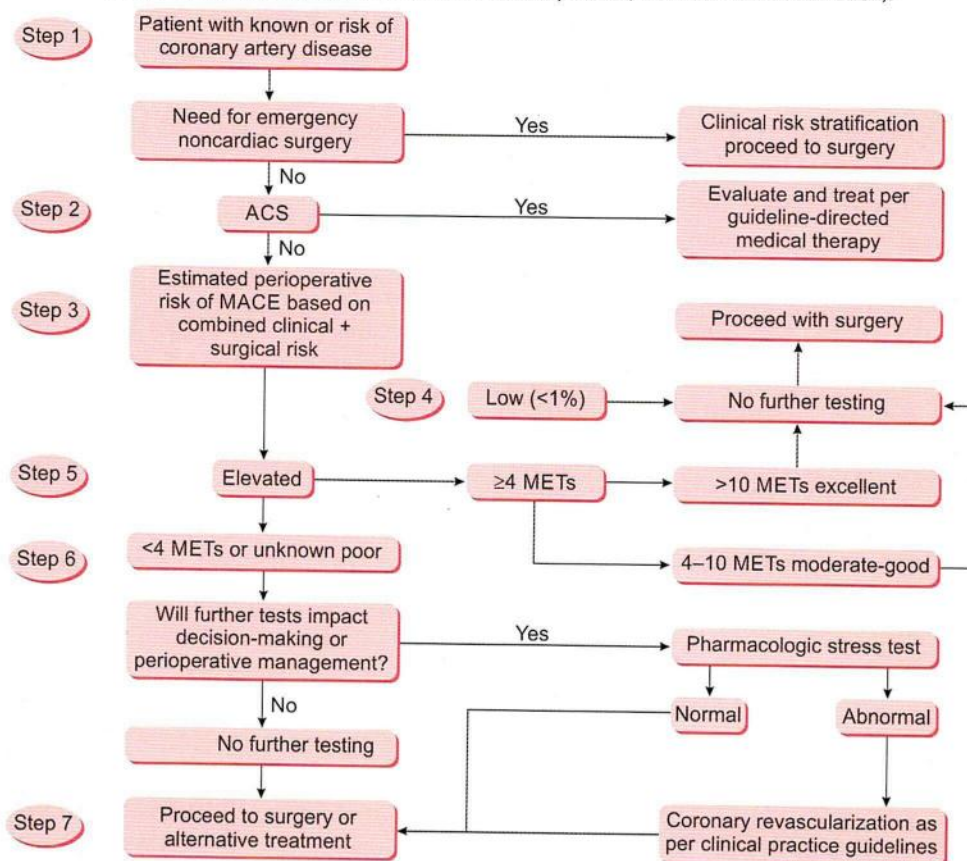
Patients with stable CAD should not undergo preoperative prophylactic coronary revascularization [based on the results of the CARP (Coronary Artery Revascularization Prophylaxis) trial].

Management of chronic medications and smoking before noncardiac surgery:

- *Aspirin:* Withhold at least 3 days before surgery and restart ASA when the risk of bleeding related to surgery has passed (i.e., 8–10 days after major noncardiac surgery) [Pulmonary Embolism Prevention (PEP) and POISE 2 trial].
- *β -blockers:* Continue the β -blocker during the perioperative period; however, if a patient's systolic BP is low before surgery, consider decreasing or holding the dose of the β -blocker before surgery (POISE trial).
- *Angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blocker (ARB):* Withhold ACE inhibitor and ARB 24 hours before noncardiac surgery and restart ACE inhibitor and ARB on day 2 after surgery, if the patient is hemodynamically stable.
- *Statins:* In patients on chronic statin therapy, continue the statin during the perioperative period.
- *Smoking:* Discuss and facilitate smoking cessation (e.g., nicotine replacement therapy), ideally starting at least 4 weeks before surgery.

Q. Summarize the plan for evaluation of a patient with IHD undergoing noncardiac surgery.

Ans. A stepwise evidence-based approach suggested by the AHA/ACC task force¹ is reproduced in **Flowchart 1**. It takes into account the urgency of surgery, the estimated risk of MACE, and the functional capacity of the patient.

Flowchart 1: Evaluation of ischemic heart disease patient (American Heart Association).

Step 1: Assess risk of CAD

Step 2: Evaluate for acute coronary syndrome (ACS)

Step 3: Calculating risk for MACE

Step 4: Low risk of MACE—proceed for surgery

Step 5: Elevated risk of MACE—assess functional status

Step 6: Whether further testing in poor functional capacity (<4 METs) impact decision-making/perioperative care

Step 7: Proceed for surgery as per guideline-directed medical therapy or alternative strategies.

Q. Which patient should be subjected to further noninvasive testing?

Ans. A patient in whom any of the following two are present should undergo further noninvasive testing:

- Intermediate clinical predictors
- Poor functional capacity (<4 METs)
- Undergoing a high-risk procedure. This could include:
 - Tests for resting LVF:
 - Current or poorly controlled CCF
 - Prior LVF and dyspnea of unknown origin
 - Exercise or pharmacologic stress test:
 - Active cardiac conditions
 - Patients with elevated risk and poor or unknown functional capacity (<4 METs).

Testing may be omitted in patients with elevated risk but with moderate or good functional capacity.

Noninvasive testing is not indicated for:

- Low-risk procedures
- No risk factors and undergoing intermediate surgery.

Q. What does one understand from the terms level of evidence (LOE) and class of recommendation (COR)?

Ans. Both help to understand the certainty of treatment effect.

Level of evidence:

- Level A: Data from multiple randomized control trials (RCTs) or meta-analyses
- Level B: Data from single RCT or nonrandomized studies
- Level C: Consensus opinion of experts, case studies, or standard of care.

Class of recommendation:

- Class I: Benefit >>> Risk (Procedure/treatment should be performed)

- **Class II:**
 - *Ila:* Benefit » Risk (Procedure/treatment *reasonable* to be performed)
 - *Iib:* Benefit ≥ Risk (Procedure/treatment *may be considered*)
- **Class III:** No benefit/harm (Procedure/treatment should not be performed).

Q. Is it necessary to subject all patients with known IHD to coronary revascularization prior to noncardiac surgery in order to reduce the risk of MACE?

Ans. According to the AHA 2014¹ and the Canadian Cardiovascular Society guidelines,⁵ revascularization before noncardiac surgery is recommended only when indicated by clinical practice guideline (IC). Routine revascularization for noncardiac surgery is not recommended (III B).

Coronary Artery Bypass Grafting

Older studies had shown a significant improvement in survival of revascularized patients, when they undergo subsequent noncardiac vascular surgery. However, the coronary artery revascularization prophylaxis (CARP) trial by McFalls et al. failed to show any benefit of coronary artery revascularization before elective vascular surgery among patients with stable cardiac symptoms.

Preoperative CABG or revascularization should be reserved for patients with:

- Left main disease
- A left ventricular ejection fraction (LVEF) <20%
- Severe aortic stenosis (AS).

For major noncardiac surgery following recent CABG, one should keep a gap of at least 4–6 weeks possibly for even up to 6 months.

Percutaneous coronary intervention (PCI): PCI before noncardiac surgery has not been shown to be of value.

Percutaneous coronary intervention before noncardiac surgery should be limited to those with:

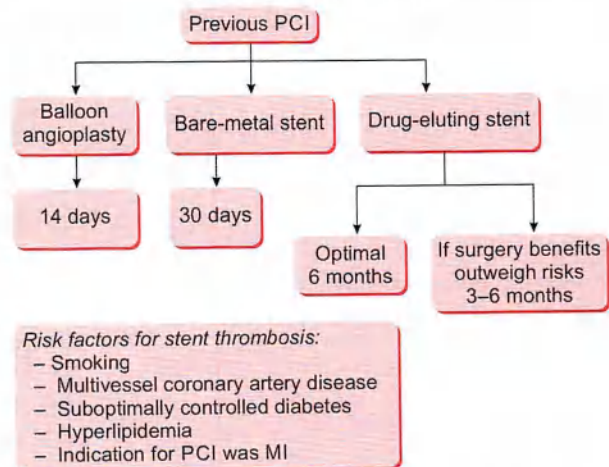
- Left main disease
- Unstable angina and MI
- Life-threatening arrhythmias due to active ischemia and could be managed with PCI.

Patients who require urgent noncardiac surgery but need prior revascularization should undergo a balloon angioplasty or bare-metal stent (BMS) implantation (**Flowchart 2**).

If PCI is necessary, then the urgency of the noncardiac surgery with the risk of bleeding and events such as stent thrombosis in a patient on dual antiplatelet therapy (DAPT) must be considered carefully. Ideally, patients have their noncardiac surgery delayed until the risks of stopping

the DAPT outweighed the risk associated with delay of noncardiac surgery.⁵

Flowchart 2: Minimum duration of delay for noncardiac surgery in a patient⁶ with previous PCI.



(PCI: percutaneous coronary intervention; MI: myocardial infarction).

Q. When can a patient who has undergone a recent coronary revascularization procedure be subjected to a noncardiac surgery?

Ans. Unscheduled noncardiac surgery in a patient who has undergone a prior PCI presents special challenges. All patients with stents require DAPT with aspirin and clopidogrel for a variable period of time. Any surgery performed within 4–6 weeks of a PTCA presents an increased risk of stent thrombosis and reinfarction if the antiplatelet medication is stopped or a risk of major bleeding if the antiplatelet treatment is continued through the perioperative period.

The 2014 ACC/AHA guidelines¹ recommended a delay of 4–6 weeks between implantation of a BMS and over a year for drug-eluting stent (DES) prior to noncardiac surgery, allowing antiplatelet therapy during stent re-endothelialization without the risk of stent blockage. These old guidelines were influenced by the fact that most studies were on first-generation DESs (paclitaxel- and sirolimus-eluting stents) which are no longer used. Studies with newer-generation DESs (everolimus- and zotarolimus-eluting stents) suggest that the risk of stent thrombosis plateaus earlier than 6–12 months after PCI. In 2016, the American College of Cardiology and the AHA have accordingly revised their recommendations⁶ (**Flowchart 2**).

Noncardiac surgery should be delayed after PCI:

- For 14 days following a balloon angioplasty in whom aspirin will need to be discontinued perioperatively

- For 30 days after a BMS
- For a period of 6 months after a DES placement
- However, noncardiac surgery may be performed 3–6 months after DES placement where the benefits of surgery are greater than the risks of stent thrombosis. The risk factors that should be factored into this decision-making include smoking, multivessel CAD, suboptimally controlled diabetes, and hyperlipidemia.
- In those patients where the PCI was performed following an MI, noncardiac surgery should be delayed for a period of 6 months irrespective of the stent type.⁷

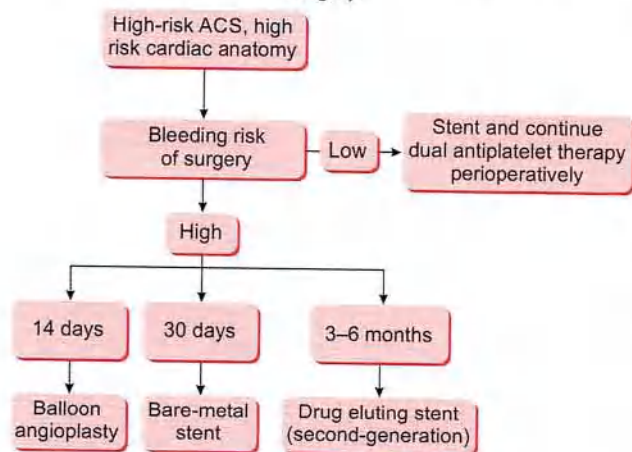
Approach as suggested in the 2016 revised guidelines by the AHA/ACC Task Force⁶ for the management of a patient who has undergone revascularization and would require urgent noncardiac surgery.

Q. Depending on the time frame available to optimize a patient prior to noncardiac surgery, which revascularization procedure would you subject the patient to?

Ans. The AHA 2014 guidelines¹ and the Canadian Cardiovascular Society Guidelines⁵ do not recommend preoperative prophylactic revascularization prior to noncardiac surgery solely to reduce the incidence of MACE in patients with stable CAD.

Management of patients requiring revascularization [as per clinical practice guidelines (CPGs)] prior to noncardiac surgery is as shown in **Flowchart 3**.

Flowchart 3: Plan for revascularization prior to urgent noncardiac surgery.



(ACS: acute coronary syndrome).

Q. Discuss the use of β -blockers to reduce perioperative cardiovascular complications along with evidence for the same.

Ans. *Rationale for the use of perioperative β -blockade therapy:* Perioperative period is a period of physiological stress associated with high levels of catecholamines

in blood. Catecholamines increase each of the four determinants of myocardial oxygen consumption (i.e., heart rate, preload, afterload, and contractility). β -blockers have the potential of reducing myocardial O_2 consumption (thus improving the myocardial O_2 supply/demand balance) by decreasing sympathetic tone and myocardial contractility, which in turn, decreases the heart rate and the arterial pressure. Mangano et al. performed a randomized, double-blind, placebo-controlled trial with atenolol in 200 patients with known CAD or risk factors for atherosclerosis who underwent noncardiac surgery. No perioperative deaths occurred in patients who were given atenolol, and only one death was reported in patients receiving the placebo. However, by 6 months, there were eight deaths in the group that received the placebo and none in the group that was given atenolol ($p < 0.001$). This difference was sustained for the 2-year follow-up period.

However, several other recent trials have not shown any benefit using perioperative β -blockade. The POISE trial found that while the incidence of cardiac complications was reduced by metoprolol, the overall mortality and the incidence of stroke were significantly higher in patients receiving metoprolol. This may have been due to a higher incidence of hypotension in this set of patients. In the POISE trial, a higher dose of a long-acting β -blocker was started immediately before the noncardiac surgery. Subsequently, three studies,^{2,8,9} where a lower dose of β -blocker was used, showed no harm or benefit.

Evidence supports the fact that the use of β -blockers can reduce perioperative cardiac events. Benefit however may be offset by a higher risk of perioperative strokes and uncertain mortality benefit.

The 2014 ACC/AHA guideline summarizes the recommendations on perioperative β -blockade¹⁰ (LOE) as follows:

Class I: Patients on β -blockers should continue their use in the perioperative period (I B).

Class II:

- β -blockers can be started preoperatively in patients with:
 - Intermediate or high-risk preoperative tests (IIB C)
 - Those with ≥ 3 RCRI factors (IIB B)
- The usefulness of β -blockers is uncertain in patients
- With long-term indication for β -blockers but no other Revised Cardiac Risk Index risk factor (IIB B):
 - Preoperative β -blockers where indicated should be started well ahead preferably >1 day prior to the surgery to assess for safety and tolerability (IIB B)
 - Restarting/modifying dose of β -blockers after surgery to be guided by clinical circumstances, independent of when the β -blocker was started (IIa B).

Class III: β -blockers are not to be started on the day of surgery (III B).

Starting β -blockers on the day of surgery may in fact be harmful. It is preferred to start the therapy at least 2–7 days prior to the surgery.

The Canadian Cardiovascular Society Guidelines⁵ too recommend against the initiation of β -blockers within 24 hours of noncardiac surgery (strong re-recommendation: high-quality evidence).

Q. What is the role of α_2 -adrenergic agonists?

Ans. Not everyone with an increased perioperative cardiac risk can tolerate a β -blocker. Clonidine, a centrally acting α_2 -adrenergic agonist, may prove to be an effective alternative. Perioperatively, it is used as a sedative, an anxiolytic, and an analgesic. It reduces hypertension, tachycardia, and norepinephrine release associated with surgical stress.

The effect of α_2 -adrenergic agonists has also been studied in the perioperative period. A meta-analysis¹¹ of 31 trials involving 4,578 patients demonstrated that α_2 -adrenergic agonists reduced death and myocardial ischemia overall but sudden discontinuation was found to be associated with hypertension, headache, agitation, and tremor. However, the POISE-2 trial which enrolled patients with or at a risk for atherosclerotic disease undergoing noncardiac surgery had a different finding. Here, clonidine was not found to reduce the rate of death or nonfatal MI, but it did increase the rate of nonfatal cardiac arrest and clinically important hypotension.

At present AHA guidelines and Canadian Cardiovascular Guidelines⁵ do not recommend the use of α_2 -adrenergic agonists for the prevention of cardiac events (III B).

Q. What about the use of statins in the perioperative period?

Ans. Statins are lipid-lowering drugs. In addition to plaque stabilization, they have been found to have anti-inflammatory and antioxidative properties, improved endothelial function, and increased nitric oxide bioavailability.

They are found to be very effective in primary and secondary prevention of cardiac events in high-risk patients.¹²

The LOAD (Lowering the Risk of Operative Complications Using Atorvastatin Loading Dose) trial,¹³ a large RCT of perioperative statin initiation found no significant difference in outcomes.

The AHA guidelines recommend: Continuation of statins in the perioperative period in those who are on chronic statin therapy (I B).

Q. Should all cardiac medications be continued throughout the perioperative period?

Ans. Patients with a history of IHD are usually on medications intended to decrease myocardial oxygen demand by decreasing the heart rate, preload, afterload, or contractile state (e.g., β -blockers, calcium channel antagonists, and nitrates) and to increase the oxygen supply by causing coronary vasodilatation (nitrates). These drugs are generally continued throughout the perioperative period. Abrupt withdrawal of β -blockers can cause rebound increases in the heart rate and the BP.

Antagonists of the renin-angiotensin-aldosterone system may occasionally cause refractory hypotension that responds only to vasopressin or terlipressin. Based on evidence from RCTs,^{14,15} previous recommendations have suggested that ARBs or ACE inhibitors be discontinued on the morning of surgery.

However, the AHA guidelines (2014)¹ suggest:

- Continuation of ACE inhibitor and ARBs in the perioperative period (IIa B)
- If withheld prior to surgery, restart as soon as clinically feasible postoperatively (IIa C).

The Canadian Cardiovascular Society⁵ however recommends withholding ACE inhibitor/Angiotensin II receptor blockers 24 hours prior to noncardiac surgery (strong recommendation: low quality evidence). As the risk of hypotension is greatest within 24 hours after surgery, they recommend starting ARB/ACE inhibitor on day 2 in those on chronic therapy provided, the patient is hemodynamically stable.

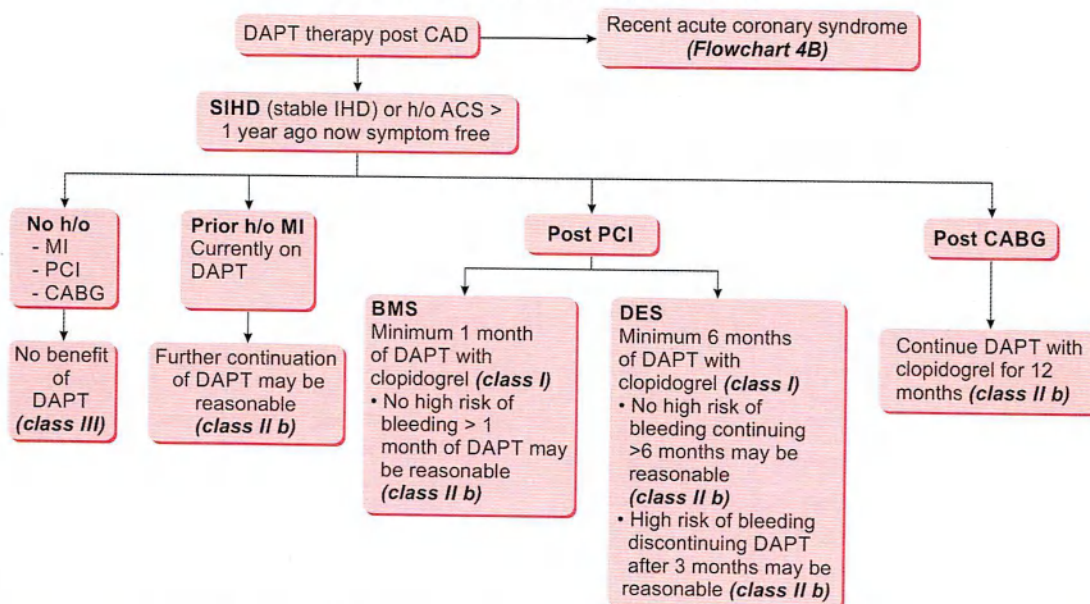
Q. How would you manage a patient on DAPT if he/she presents for a noncardiac surgery?

Ans. All patients with stents require DAPT with aspirin and clopidogrel for a variable period of time. These patients present a challenge to the anesthetist. The risk of bleeding exists with the continuation of DAPT while discontinuing the antiplatelet therapy runs the risk of stent thrombosis and reinfarction. The current AHA/ACC guidelines⁶ for the management of a patient on DAPT are shown in **Flowcharts 4A and B**.

Q. How long should a patient with a recent MI wait before undergoing elective noncardiac surgery?

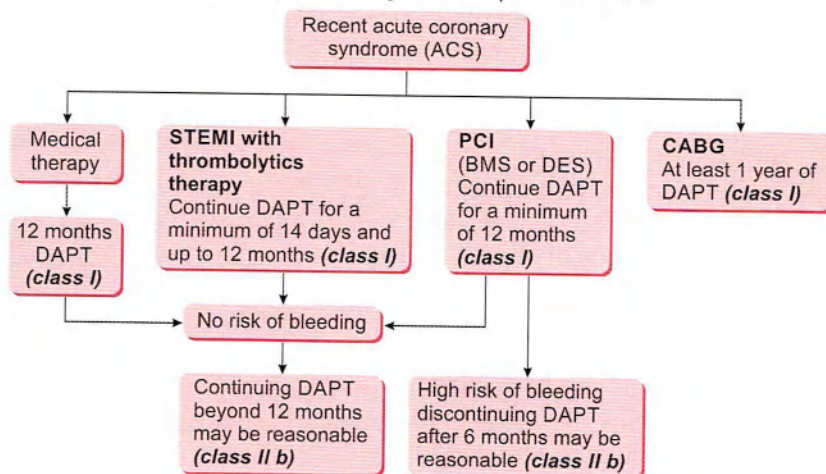
Ans. The risk of reinfarction during surgery after a prior MI has traditionally depended on the time interval between the MI and the procedure. Postoperative MI and 30-day mortality rates are known to decrease substantially as the length of time from the MI to the operation is increased. The risk modified further by coronary interventions at the time of the MI. In the past, ACC/AHA task force suggested that elective surgery is associated with prohibitive risk

Flowchart 4A: Management of a patient on DAPT.



(ACS: acute coronary syndrome; BMS: bare-metal stent; CABG: coronary artery bypass grafting; CAD: coronary artery disease; DAPT: dual antiplatelet therapy; DES: drug-eluting stent; IHD: ischemic heart disease; MI: myocardial infarction; PCI: percutaneous coronary intervention).

Flowchart 4B: Management of a patient on DAPT.



(BMS: bare-metal stent; CABG: coronary artery bypass grafting; DAPT: dual antiplatelet therapy; DES: drug-eluting stent; STEMI: ST-elevation myocardial infarction; PCI: percutaneous coronary intervention).

only during the initial 4–6 weeks. However, the AHA 2014 guidelines suggest that noncardiac surgery in the absence of any coronary intervention could be performed ≥ 60 days after an MI.

A recent MI, defined as having occurred within 6 months of noncardiac surgery, is considered as an independent risk factor for perioperative stroke, with an eightfold increase in the perioperative mortality rate.¹⁶

Q. How is premedication useful in the setting of IHD and surgery?

Ans. Patient anxiety can lead to catecholamine secretion and increased oxygen demand. In this regard, the goal of premedication is to produce sedation and amnesia without causing deleterious myocardial depression, hypotension, or hypoxemia. Morphine, scopolamine, and benzodiazepines, alone or in combination, are popular choices to achieve

these goals. All premedicated patients should receive supplemental oxygen.

Q. Outline the hemodynamic goals of induction and maintenance of general anesthesia in patients with IHD.

Ans. The anesthesiologist's goal must be to maintain the balance between myocardial O₂ demand and supply throughout the perioperative period. During induction, wide swings in heart rate and BP should be avoided. Ketamine should be avoided because of the resultant tachycardia and hypertension. Prolonged laryngoscopy should be avoided, and the anesthesiologist may wish to blunt the stimulation of laryngoscopy and intubation by the addition of opiates, β -blockers, or laryngotracheal or intravenous (IV) lignocaine.

Maintenance drugs are chosen with knowledge of the patient's ventricular function. In patients with good LVEF, the cardiac depressant and vasodilatory effects of inhaled anesthetics may reduce myocardial O₂ demand. An opioid-based technique may be chosen to avoid undue myocardial depression in patients with poor LVEF. Muscle relaxants with minimal cardiovascular effects are usually preferred.

Blood pressure and heart rate should be maintained near baseline values. This can be accomplished by blunting sympathetic stimulation with adequate analgesia and aggressively treating hypertension (e.g., anesthetics, nitroglycerin, nitroprusside, and β -blockers), hypotension (e.g., fluids, sympathomimetics, and inotropic drugs), and tachycardia (e.g., fluids, anesthetics, and β -blockers).

Q. Which monitors are useful for detecting ischemia intraoperatively?

Ans. The following monitors are useful for detecting perioperative ischemia:¹⁷

Electrocardiogram: In the perioperative period, ECG monitoring most commonly helps to identify stress-induced ST depression type of ischemia. Most current monitors do not simultaneously allow monitoring of 12-ECG leads. Selecting which chest leads to monitor is important particularly in noncardiac surgery. The combination of various leads with their sensitivity for detecting PMI is as given below:

- The V₅ (Sensitivity: 75%) is the most sensitive single ECG lead for detecting ischemia and should be monitored routinely.
- Lead V₄ (61%)
- Leads V₄ and V₅ (90%)
- Leads II and V₅ (80%)
- Leads II, V₄, and V₅ (98%).

Transesophageal echocardiography (TEE): It can provide continuous intraoperative monitoring of LVEF. Detection

of regional wall motion abnormalities with this technique is the most sensitive monitor for myocardial ischemia. However, according to the AHA 2014 guidelines:

- Routine use of intraoperative TEE in noncardiac surgery is not recommended (III C)
- Emergency use of TEE in patients with hemodynamic instability is reasonable only if experts are readily available (IIa C).

Pulmonary artery catheters: Pulmonary artery occlusion (wedge) pressure gives an indirect measurement of left ventricular volume and is a useful guide to optimizing intravascular fluid therapy. Sudden increases in the wedge pressure may indicate acute left ventricular dysfunction due to ischemia. As per AHA 2014 guidelines:

- The routine use of pulmonary artery catheters in patients with IHD has not been shown to improve outcome and hence is not recommended (III A).
- Pulmonary artery catheters may be considered in patients in whom the underlying medical conditions that significantly affect hemodynamics cannot be corrected preoperatively (IIb C).

Q. What are the goals of anesthetic management in this patient?

Ans. The primary goal of anesthetic management in a patient with IHD for noncardiac surgery is the avoidance of myocardial ischemia and MI. This is accomplished by preventing ischemia through measures that improve the myocardial oxygen supply-demand balance, primarily by controlling the patient's hemodynamics, and by detecting and treating myocardial ischemia when and if it does occur. The pathophysiology of PMI differs somewhat from that of MI occurring in the nonoperated patient, where rupture of a coronary arterial atherosclerotic plaque leads to platelet aggregation and thrombus formation (Type I MI). In contrast, plaque rupture is not always the cause of PMIs; most MIs which occur perioperatively are due to a prolonged imbalance between myocardial oxygen supply and demand in the setting of CAD (Type II MI).¹⁷

Myocardial Oxygen Supply-Demand Balance

The myocardium extracts most of the oxygen from the blood flowing through the coronary arteries in a normal individual at rest. Increased cardiac work for any reason (e.g., physical exertion, emotional stress, and hypertension) increases myocardial oxygen demand, which is compensated in a healthy person by an increased CBF. With IHD, the arteries are narrowed and the CBF is already maximal. When oxygen demand by the myocardium exceeds its oxygen supply, myocardial ischemia develops, which, if prolonged, results in MI.

TABLE 3: Causes of imbalance in myocardial oxygen supply and demand.

Decreased oxygen supply	Increased oxygen demand
Decreased coronary blood flow • Tachycardia (diastolic perfusion time) • Hypotension (especially diastolic) • Increased preload (perfusion pressure) • Hypocapnia (coronary vasoconstriction) • Coronary artery spasm	Tachycardia • Increased wall tension • Increased preload • Increased afterload (hypertension) • Increased myocardial contractility
Decreased oxygen content and availability • Anemia • Hypoxemia • Reduced oxygen release from hemoglobin (e.g., pH, 2,3-DPG, and temperature)	

Hence, the cornerstone of prevention and treatment of myocardial ischemia is to improve oxygen supply and reduce oxygen demand. It is important to know the conditions detrimental to myocardial oxygen balance because they are encountered frequently in the perioperative period, and can be caused and/or manipulated to some degree by the anesthesiologist (Table 3).

Q. Describe perioperative anesthetic management for the above patient?

Ans. All anesthetic techniques must aim to keep myocardial oxygen supply greater than demand, and therefore avoid ischemia. The essential requirements of general anesthesia for a patient with IHD are avoiding tachycardia and extremes of BP, both of which adversely affect the balance between oxygen supply and demand.

Premedication: An anxious patient may be tachycardic and require an anxiolytic premedication. The patient may already be on medication for angina or hypertension. These drugs include β -blockers, nitrates, and calcium antagonists. These protect against the hemodynamic stresses of surgery and should be continued through the perioperative period. However, general anesthesia may exaggerate the hypotensive actions of such drugs.

Induction: All IV anesthetic agents have a direct depressant action on the myocardium, and may also reduce vascular tone. This causes hypotension (especially in the hypovolemic patient), often with a compensatory tachycardia, which may cause myocardial ischemia. To avoid hypotension at the time of induction, all agents should be given slowly and in small increments. However, since ketamine causes indirect stimulation of the sympathetic nervous system, leading to both hypertension (increased afterload) and tachycardia, it should be avoided.

Intubation: Laryngoscopy is a powerful stressor, causing hypertension and tachycardia. This can be avoided with a supplemental dose of IV induction agent or liberal dose of an opioid such as fentanyl prior to laryngoscopy. Also, minimize duration of laryngoscopy (15 seconds or less) which may be useful in reducing the magnitude of sympathetic stimulation.

Maintenance: Volatile agents have minimal effects on cardiac output, although they do reduce myocardial contractility, especially halothane. They cause vasodilatation, and isoflurane has been implicated in the “coronary steal” syndrome. The theory is that prestenotic vasodilatation diverts blood away from already ischemic areas of the myocardium. However, there is doubt as to the clinical significance of this phenomenon. Vagal stimulation due to halothane can cause bradycardias and nodal rhythms. Bradycardias can be beneficial by allowing greater coronary diastolic filling, provided BP is maintained.

Iatrogenic hyperventilation, which greatly decreases PaCO_2 , should be avoided as hypocapnia may evoke coronary artery vasoconstriction. It is important to avoid persistent and excessive changes in the heart rate and systemic BP. A common recommendation is to strive to maintain patient's heart rate and BP within 20% of normal awake value. Maintain good pain relief and adequate depth of anesthesia to minimize sympathoadrenal response. Pain relief should be provided with liberal use of opioids. Theoretically, nonsteroidal anti-inflammatory drugs (NSAIDs) may have both a useful postoperative analgesic action and an antiplatelet effect which may reduce coronary thrombosis.

Maintain normothermia during intraoperative period as hypothermia will lead to peripheral vasoconstriction, thus leading to increased afterload. Also, these patients will develop shivering in postoperative period thus increasing myocardial work and oxygen demand.

Anemia is well-tolerated in the general population, but can cause a critical reduction in myocardial oxygen supply in those with IHD—a hematocrit of 30% or more is recommended. The American Association of Blood Banks 2012 Clinical Practice Guidelines¹⁸ recommend:

- **Transfusion in patients with CAD if:**
 - Hemoglobin is <8 g/dL
 - Patient is symptomatic for chest pain, orthostasis, and CHF
- In postoperative patients, it is recommended to maintain hemoglobin level of ≥ 8 g/dL unless patient exhibits symptoms mentioned above.

As per the AHA 2014 guidelines:

- It is reasonable to use either a volatile anesthetic or total intravenous anesthesia (TIVA) in noncardiac surgery (IIa A).

- Maintenance of normothermia may be reasonable to prevent perioperative cardiac events (IIb B).
- Use of IV nitroglycerine (NTG) prophylactically is not effective in reducing the incidence of perioperative myocardial ischemia (III B).

Reversal and recovery: Reversal of muscle relaxation with a combined anticholinesterase/antimuscarinic causes tachycardia, and extubation in itself is a stressor. Problems in the recovery phase which can cause ischemia include tachycardia, pain, hypothermia, shivering, hypoxia, and anemia. These should be treated not just in the immediate postoperative period, but throughout the hospital admission. The use of supplemental oxygen in the postoperative period is one of the simplest, yet most effective measures in preventing myocardial ischemia.

Neuraxial anesthesia and perioperative pain management: The use of regional anesthetic techniques has theoretical advantages—epidural anesthesia reduces preload and afterload, reduces coagulation responses, and, in the case of thoracic epidurals, causes coronary vasodilatation. These effects should reduce perioperative myocardial ischemia, but this is not supported by any evidence. However, good epidural analgesia may reduce the incidence of tachycardia arising due to postoperative pain. However, care should be taken to optimally fill these patients before giving a central neuraxial blockade. Despite the decrease in myocardial O₂ requirements produced by peripheral sympathetic nervous system blockade, it is important to realize that flow in the coronaries narrowed by atherosclerosis is pressure dependent. Therefore, decreases in systemic BP with epidural or spinal anesthesia should not be permitted to persist. In a patient with IHD, local anesthetic techniques whenever feasible such as brachial plexus block should be encouraged in order that the hemodynamic responses to general anesthesia are avoided. However, even under local anesthesia, the patient will be subject to the stresses of the surgical procedure itself as well as anxiety related to it, which can have marked hemodynamic effects.

Recent evidence^{19,20} have shown that intraoperative neuraxial anesthetic does not provide any cardioprotective benefit over general anesthesia. However, the current AHA 2014 guidelines suggest the use of:

- Neuraxial anesthesia for postoperative pain relief to decrease the incidence of PMI in patients undergoing abdominal aortic surgery (IIa B)
- Preoperative epidural analgesia may be considered to decrease the incidence of preoperative cardiac events in patients with hip fractures (IIb B).

Q. Describe your strategies to prevent PMI in this patient.

Ans. The risk of PMI peaks within first 72 hours after surgery with most events occurring on the day of surgery or next

24 hours. PMI is due to either acute plaque disruption (Type I MI) or due to a myocardial oxygen supply/demand mismatch (Type II MI). Increases in sympathetic discharge with accompanying elevation of heart rate and BP, procoagulant postoperative environment, and mobilization of interstitial fluid administered during perioperative period may promote plaque rupture and subsequent cardiac event.

Strategies to prevent PMI would therefore include:

- Optimization as per the current AHA guidelines as mentioned above
- Continuation of appropriate pharmacotherapy perioperatively
- Maintain a supply-demand balance.

Q. What postoperative care would you advice for this patient?

Ans.

- Continuous ECG monitoring and surveillance for postoperative myocardial ischemia and infarction
- Continuation or institution of β -blockade guided by clinical parameters
- **Temperature control:** Decreases in body temperature that occur intraoperatively may predispose to shivering on awakening leading to abrupt excessive increases in myocardial O₂ requirement
- Provision of supplemental O₂
- Adequate postoperative pain relief
- Maintenance of hemodynamic parameters with IV fluids
- Deep venous thrombosis prophylaxis.

Q. How would you diagnose and manage PMI in this patient?

Ans. Mechanisms of PMI: The risk of PMI peaks within first 72 hours after surgery with most events occurring on the day of surgery or next 24 hours. Most of these events are silent and of non-ST-elevation myocardial infarction (NSTEMI) variety, as shown by various studies. In PMI, acute plaque disruption and hemorrhage in the infarct-related coronary artery seem to be common. On the other hand, a myocardial oxygen supply/demand mismatch is often believed to be the main trigger of myocardial injury. However, myocardial oxygen supply/demand mismatch and plaque rupture are not mutually exclusive mechanisms, and MIs may develop by different mechanisms at different locations in the same patient. In cases of fatal PMIs, autopsy findings have shown plaque rupture as the cause of PMI in 45–55% cases, whereas half of the patients had no evidence of plaque rupture in their coronaries in spite of having extensive CAD. Increases in sympathetic discharge with accompanying elevation of heart rate and BP, procoagulant postoperative environment, and mobilization of interstitial fluid administered during perioperative period may promote plaque rupture and subsequent cardiac event.

Most PMIs (>80%) which occur early after surgery, are asymptomatic, are of the non-Q-wave type and are most commonly preceded by ST-segment depression rather than ST-segment elevation. The majority of PMIs is silent showing no signs or symptoms and may be completely overlooked if continuous ECG monitoring is not performed. Long duration (single duration >20–30 minutes or cumulative duration >1–2 hours) rather than merely the presence of postoperative ST-segment depression seems to be the important factor associated with adverse cardiac outcome. The frequent combination of increases in heart rate preceding the ischemic episodes, ST-segment depression rather than elevation during all ischemic episodes, non-Q-wave rather than Q-wave MIs in almost all cases, the lack of angiographically visible thrombus or ruptured plaques in some patients who underwent coronary angiography following PMI, and complete reversal of ECG changes to baseline are highly suggestive that prolonged stress-induced myocardial ischemia is the likely primary cause of PMI. Repeated brief ischemic episodes may well have a cumulative effect and ultimately cause myocardial necrosis.

In summary, it can be said that both plaque rupture and prolonged perioperative ischemia in presence of stable CAD contribute to PMI. Probably, this could explain the benefit of β -blockade therapy.

Diagnosis

According to the definition of the World Health Organization (WHO), at least two of three criteria must be fulfilled to diagnose MI: (1) typical ischemic chest pain; (2) increased serum concentration of creatine kinase (CK)-MB isoenzyme and troponin; and (3) typical electrocardiographic findings, including development of pathological Q waves.

Perioperative myocardial infarction is mostly silent. Most MIs occur within 48 hours after noncardiac surgery when analgesics that the patient receives can mask ischemic symptoms and are associated with an increased risk of 30-day mortality. In the past, studies have shown that ST-segment changes postoperatively were associated with an increased incidence in MACE. However, recent evidence has suggested that an ECG may not provide sufficient information in this setting. Even in nonsurgical patients, ECG is diagnostic only in 50% of cases, some abnormality but not classic MI changes in 40% and normal in 10% patients. Also, pain if present is often masked by analgesia and residual anesthetics in the immediate perioperative period. Therefore, if the diagnosis of MI is based solely on the classical triad, considerable underreporting could happen.

Routine ICU monitoring with two-lead ECG and ST-segment trending detects ischemia only in 3% of high-risk postoperative patients when compared to 12-lead ECG.⁹ The

majority of ischemic events occurred in leads V₁, V₂, and V₄ and not in commonly monitored leads, II and V₅. Also, preexisting bundle branch blocks, digitalis effects, or paced beats make interpretation of ECG difficult.

All these limitations put heavy emphasis on biochemical markers as a diagnostic test in perioperative period. CK-MB is the most routinely available and done marker and has sensitivity of 60–75% and specificity of 80–95% in the perioperative period.

The *cardiac troponins* (troponins T and I) are cardiac injury markers that are rapidly released into the circulation after myocyte injury. Troponins have nearly absolute myocardial tissue specificity and a high sensitivity. Several large studies have demonstrated the superiority of troponin testing to ECG in identifying patients with type I and type II MI, and suggest that it may be superior initial test in the diagnosis of MI. An elevated troponin level has been found to be the strongest predictor of 30-day mortality²¹ and an independent predictor of all-cause mortality.²²

Myocardial injury after noncardiac surgery (MINS) was defined as a peak fourth-generation troponin T ≥ 0.03 ng/mL believed to be due to myocardial ischemia. Most of these events were asymptomatic and detected only through routine surveillance of postoperative troponin levels.²³

Additional testing with *ECHO* to assess LVF may be required to establish definite diagnosis with the detection of a new regional wall motion abnormality (RWMA).

The recent AHA guidelines recommend that:

- Troponin levels should be measured in the setting of signs and symptoms suggestive of MI (I A).
- ECG should be recorded in the setting of signs and symptoms suggestive of MI (I B).
- Usefulness of postoperative screening with either troponins or ECG in patients with high risk for PMI but without signs and symptoms is uncertain (IIb B).
- Routine postoperative screening in unselected patients without any signs and symptoms of MI or ischemia is not useful for guiding perioperative management (III B).

Management of PMI

The major difference between perioperative patients and nonsurgical patients is the risk of life-threatening bleeding with thrombolytic therapy in the former. Thus, thrombolysis is almost always contraindicated. The bleeding risk is high even with aggressive use of antiplatelet agents and anticoagulants. Hence, in the perioperative setting, a more conservative approach is recommended. Urgent angiography and PCIs are reserved for patients with STEMI or those with NSTEMI who are hemodynamically unstable. The mainstay of management is the medical line of treatment consisting of good pain control, β -blockade, antiplatelet agents, and anticoagulants:

- **Oxygen:** It should be supplemented in patients with an arterial saturation <90%, patients in respiratory distress, or those with other high-risk features for hypoxemia.²⁴
- **Pain relief:** Pain if present should be treated with opioids, most commonly used being morphine, as it decreases preload, thus decreasing myocardial oxygen consumption.
- **Nitrates:** As certain studies did not show survival benefit for nitrates, use of nitrates is optional for patients without evidence of ongoing ischemia, but for patients with symptomatic ischemia, IV NTG is effective.
- **Antiplatelet agents:** Aspirin should be administered as soon as MI is suspected in the dose of 375 mg orally through NGT. Other antiplatelet agents are available, clopidogrel being the widely used one. In patients with allergy to aspirin, clopidogrel can be used. Aspirin being a weak antiplatelet agent, it is advisable to combine both; however, studies have shown that combination increases the relative risk for major perioperative bleeding by approximately 50%.
- **Anticoagulants:** Acute MI is often associated with increased thrombin activity. Unfractionated heparin is associated with indirect thrombin inhibition and is frequently used to treat acute MI. Low molecular weight heparins act through their anti-Xa activity and have predictable kinetics but suffer from the longer half-life. In the perioperative setting, IV UFH is indicated if the suspicion of plaque rupture is high and bleeding risk is low.
- **β -blockers:** β -blockers should be used for all the patients with acute coronary event unless there is significant bradycardia, decompensated CHF, or severe COPD. Calcium channel blockers have no survival benefit in such situations and should not be used.
- **Statins:** They are currently recommended for all patients who develop PMI.

The Canadian Cardiovascular Society therefore recommends⁵ that in patients with a baseline risk >5% for cardiovascular death or nonfatal MI at 30 days after surgery, i.e., patients with the following:

- An elevated preoperative NT-proBNP/BNP
- An RCRI score ≥ 1
- Age of 45–64 years with significant cardiovascular disease
- Age of ≥ 65 years.

In the postoperative period, the following are recommended⁵ to identify myocardial injury:

- Daily troponin levels for 48–72 hours after noncardiac surgery (strong recommendation: moderate-quality evidence)
- An ECG in the postanesthesia care unit (conditional recommendation: low-quality evidence)

- Shared-care management by surgeons and other medical specialists in the care of such patients
- Against the use of pulmonary artery catheters in patients undergoing noncardiac surgery (strong recommendation: moderate-quality evidence).

In patients who suffer a myocardial injury or MI after noncardiac surgery, the Canadian Cardiovascular Society⁵ recommends:

- Initiation of long-term ASA (strong recommendation: moderate-quality evidence)
- Long-term statins (strong recommendation: moderate-quality evidence).

REFERENCES

1. Fleisher LA, Fleischmann KE, Auerbach AD, Barnason SA, Beckman JA, Bozkurt B, et al. 2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery: a report of the American College of Cardiology/American Heart Association Task Force on practice guidelines. *J Am Coll Cardiol*. 2014;64(22):e77-137.
2. Juul AB, Wetterslev J, Kofoed-Enevoldsen A, Callesen T, Jensen G, Gluud C; Diabetic Postoperative Mortality and Morbidity group. The Diabetic Postoperative Mortality and Morbidity (DIPOM) trial: rationale and design of a multicenter, randomized, placebo-controlled, clinical trial of metoprolol for patients with diabetes mellitus who are undergoing major noncardiac surgery. *Am Heart J*. 2004;147(4):677-83.
3. Lee TH, Marcantonio ER, Mangione CM, Thomas EJ, Polanczyk CA, Cook EF, et al. Derivation and prospective validation of a simple index for prediction of cardiac risk of major noncardiac surgery. *Circulation*. 1999;100(10):1043-9.
4. Gupta PK, Gupta H, Sundaram A, Kaushik M, Fang X, Miller WJ, et al. Development and validation of a risk calculator for prediction of cardiac risk after surgery. *Circulation*. 2011;124(4):381-7.
5. Ducepe E, Parlow J, MacDonald P, Lyons K, McMullen M, Srinathan S, et al. Canadian Cardiovascular Society Guidelines on Perioperative Cardiac Risk Assessment and Management for patients who undergo Noncardiac Surgery. *Can J Cardiol*. 2017;33(1):17-32.
6. Levine GN, Bates E, Bitl JA, Brindis RG, Fihn SD, Fleisher LA, et al. 2016 ACC/AHA guideline focused update on duration of dual antiplatelet therapy in patients with coronary artery disease: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines: An Update of the 2011 ACCF/AHA/SCAI Guideline for Percutaneous Coronary Intervention, 2011 ACCF/AHA Guideline for Coronary Artery Bypass Graft Surgery, 2012 ACC/AHA/ACP/AATS/PCNA/SCAI/STS Guideline for the Diagnosis and Management of Patients With Stable Ischemic Heart Disease, 2013 ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction, 2014 AHA/ACC Guideline for the Management of Patients With Non-ST-Elevation Acute Coronary Syndromes, and 2014 ACC/AHA Guideline on Perioperative Cardiovascular

- Evaluation and Management of Patients Undergoing Noncardiac Surgery. *Circulation*. 2016;134:e123-55.
7. Holcomb CN, Hollis RH, Graham LA, Richman JS, Valle JA, Itani KM, et al. Association of coronary stent indication with postoperative outcomes following noncardiac surgery. *JAMA Surg*. 2016;151(5):462-9.
8. Brady AR, Gibbs JSR, Greenhalgh RM, Powell JT, Sydes MR; POBBLE trial investigators. Perioperative beta-blockade (POBBLE) for patients undergoing infrarenal vascular surgery: results of a randomized double-blind controlled trial. *J Vasc Surg*. 2005;41(4):602-9.
9. Yang H, Raymer K, Butler R, Parlow J, Roberts R. The effects of perioperative beta-blockade: results of the Metoprolol after Vascular Surgery (MaVS) study, a randomized controlled trial. *Am Heart J*. 2006;152(5):983-90.
10. Wijeyesundera DN, Duncan D, Nkonde-Price C, Virani SS, Washam JB, Fleischmann KE, et al. Perioperative beta blockade in noncardiac surgery: a systematic review for the 2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol*. 2014;130(24):2246-64.
11. Wijeyesundera DN, Naik JS, Beattie WS. Alpha-2 adrenergic agonists to prevent perioperative cardiovascular complications: a meta-analysis. *Am J Med*. 2003;114(9):742-52.
12. Ridker PM, Wilson PW. A trial-based approach to statin guidelines. *JAMA*. 2013;310(11):1123-4.
13. Berwanger O, de Barros E Silva PG, Barbosa RR, Precoma DB, Figueiredo EL, Hajjar LA, et al. Atorvastatin for high-risk statin-naïve patients undergoing noncardiac surgery: the Lowering the Risk of Operative Complications Using Atorvastatin Loading Dose (LOAD) randomized trial. *Am Heart J*. 2017;184:88-96.
14. Rosenman DJ, McDonald FS, Ebbert JO, Erwin PJ, LaBella M, Montori VM. Clinical consequences of withholding versus administering renin-angiotensin-aldosterone system antagonists in the preoperative period. *J Hosp Med*. 2008;3(4):319-25.
15. Turan A, You J, Shiba A, Kurz A, Saager L, Sessler DI. Angiotensin converting enzyme inhibitors are not associated with respiratory complications or mortality after noncardiac surgery. *Anesth Analg*. 2012;114(3):552-60.
16. Mashour GA, Shanks AM, Kheterpal S. Perioperative stroke and associated mortality after noncardiac, nonneurologic surgery. *Anesthesiology*. 2011;114(6):1289-96.
17. Miller RD, Cohen NH, Eriksson LI, Fleisher LA, Wiener-Kronish JP, Young WL. *Miller's Anesthesia*. 8th edition. Philadelphia: Elsevier Saunders; 2015.
18. Carson JL, Grossman BJ, Kleinman S, Tinmouth AT, Marques MB, Fung MK, et al. Red blood cell transfusion: a clinical practice guideline from the AABB. *Ann Intern Med*. 2012;157(1):49-58.
19. Barbosa FT, Jucá MJ, Castro AA, Cavalcante JC. Neuraxial anaesthesia for lower-limb revascularization. *Cochrane Database Syst Rev*. 2013;(7):CD007083.
20. Norris EJ, Beattie C, Perler BA, Martinez EA, Meinert CL, Anderson GF, et al. Double-masked randomized trial comparing alternate combinations of intraoperative anesthesia and postoperative analgesia in abdominal aortic surgery. *Anesthesiology*. 2001;95(5):1054-67.
21. Devereaux PJ, Chan MT, Alonso-Coello P, Walsh M, Berwanger O, Villar JC, et al. Association between postoperative troponin levels and 30-day mortality among patients undergoing noncardiac surgery. *JAMA*. 2012;307(21):2295-304.
22. Levy M, Heels-Ansdell D, Hiralal R, Bhandari M, Guyatt G, Yusuf S, et al. Prognostic value of troponin and creatine kinase muscle and brain isoenzyme measurement after noncardiac surgery: a systematic review and meta-analysis. *Anesthesiology*. 2011;114(4):796-806.
23. Botto F, Alonso-Coello P, Chan MT, Villar JC, Xavier D, Srinathan S, et al. Myocardial injury after noncardiac surgery: a large, international, prospective cohort study establishing diagnostic criteria, characteristics, predictors, and 30-day outcomes. *Anesthesiology*. 2014;120(3):564-78.
24. Amsterdam EA, Wenger NK, Brindis RG, Casey DE Jr, Ganiats TG, Holmes DR Jr, et al. 2014 AHA/ACC guideline for the management of patients with non-ST-elevation acute coronary syndromes: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2014;130(25):2354-94.

Anesthetic Considerations for Hypertensive Patient

Vijaya Prakash Patil, Madhavi Shetmahajan, Sukhada Savarkar

A 49-year-old female with carcinoma of esophagus weighing 56 kg is posted for total esophagectomy. She is a known hypertensive and diabetic for the past 10 years, and is on tablets losartan 50 mg daily and metformin 500 mg daily in two divided doses and mixtard insulin 10 units before breakfast. On examination, she has pulse 84/min, blood pressure 140/90 mm Hg, and good effort tolerance (three flights). She has some missing teeth and also buck teeth.

Q. Define hypertension.

Ans. Hypertension is the elevation of blood pressure (BP) more than normal. Primary or essential hypertension is a contributing risk factor to adverse cardiovascular system, central nervous system, and renal events.

Several organizations have proposed guidelines on this topic with slight difference in definition, spectrum, and management of hypertension. Nevertheless, hypertension is a common comorbidity which needs to be treated as it is progressive in nature with systemic manifestations. Preoperative BPs >180/110 mm Hg require careful evaluation of the situation and should go ahead with surgery only if benefits are far more than delay for optimization. Also, in a hypertensive patient, intraoperative hemodynamic instability should be anticipated and clinician should be prepared for it.

JNC-8 (Joint National Committee) guidelines (2015):¹ According to JNC-8, hypertension is defined as high BP $\geq 140/90$ mm Hg. It recommends treatment BP goal of <150/90 mm Hg for hypertensive persons aged 60 years or older and diastolic goal of <90 mm Hg in hypertensive persons between 30 and 59 years of age with the help of lifestyle modifications (reduced salt intake, aerobic exercise, moderation in alcohol consumption, high fiber, less sugar diet, etc.) and pharmacotherapy.

The panel recommends a BP of <140/90 mm Hg for young individuals (<60 years), diabetics or nondiabetics with chronic kidney disease (CKD).

Drug treatment to be initiated with an angiotensin-converting enzyme (ACE) inhibitor, angiotensin receptor blocker (ARB), calcium channel blocker (CCB), or thiazide-type diuretic in the nonblack hypertensive population,

including those with diabetes. The β -blockers are not included in drugs for first-line therapy.

Seventh report of Joint National Committee (JNC-7) had categorized hypertension as given in **Table 1.**²

TABLE 1: Categorization of hypertension as seventh report of Joint National Committee.

Category	Blood pressure (mm Hg)
Normal	SBP (systolic blood pressure) 90–119 and DBP (diastolic blood pressure) 60–79
Prehypertension	SBP 120–139 or DBP 80–89
Stage 1 hypertension	SBP 140–159 or DBP 90–99
Stage 2 hypertension	SBP ≥ 160 or DBP ≥ 100

Recently in 2017, **American College of Cardiology/American Heart Association (ACC/AHA)**³ published guidelines for prevention, detection, evaluation, and management of high BP. It is an update of the seventh report of JNC. The guideline gives a comprehensive report of diagnosis and management of hypertension and also covers management of hypertension in adult with various comorbidities. It classifies BP into four categories as follows:

1. **Normal:** <120/<80 mm Hg
2. **Elevated:** 120–129/<80 mm Hg
3. **Stage 1 hypertension:** 130–139 or 80–89 mm Hg
4. **Stage 2 hypertension:** ≥ 140 or ≥ 90 mm Hg.

This change in the classification of BP is based on the association between hypertension and risk of cardiovascular disease (CVD). It is well known that both systolic blood pressure (SBP) and diastolic blood pressure (DBP) are associated with increased risk of CVD. Several studies also indicate that as the BP progresses from normal to elevated to stages 1 and 2; the risk of CVD increases proportionately.

The ACC/AHA 2017 guidelines emphasize on the importance of out-of-office BP and recommend that out-of-office BP measurements should be used to confirm the diagnosis of hypertension as well as to adjust the treatment of hypertension. The committee also recommends nonpharmacological therapy in elevated BP and hypertension either alone or in combination with BP-lowering medication. Nonpharmacological therapy includes weight loss for overweight or obese patients, diet modification, e.g., the DASH (Dietary Approaches to Stop Hypertension) diet, sodium restriction, potassium supplementation unless contraindicated, increased physical activity, and limited alcohol consumption. For pharmacological treatment, it recommends thiazide diuretics, CCBs, and ACE inhibitors or ARBs as first-line agents as in JNC-7 guidelines.

Pharmacological treatment is to be started for:

- BP $\geq 130/80$ mm Hg in a patient with presence of CVD or an estimated 10-year ASCVD (atherosclerotic cardiovascular disease) risk of $\geq 10\%$.
- BP $\geq 140/90$ mm Hg in a patient with no history of CVD and an estimated 10-year ASCVD risk $< 10\%$.

The treatment goal in elderly patients who are ambulatory and noninstitutionalized is SBP of < 130 mm Hg which is lower than most of the hypertension guidelines in the past.

In 2018, European Society of Cardiology/European Society of Hypertension (ESC/ESH)⁴ also published guidelines for hypertension. There are a few differences between these guidelines and 2017 ACC/AHA guidelines; but in general both guidelines recommend a lower BP target for treatment including elderly patients.

Miller defines hypertension as two or more arterial BP measurements $> 140/90$ mm Hg.

In other age group, systemic hypertension is diagnosed when the BP is above levels mentioned below:

- *Adolescent*: 100/75 mm Hg
- *Early childhood*: 85/55 mm Hg
- *Infant*: 70/45 mm Hg.

Monitoring BP in the Perioperative Period

Blood pressure can be measured directly (invasive) and indirectly (noninvasive). Standard noninvasive blood pressure (NIBP) monitoring is mandatory for anesthesia, with a minimal interval of 5 minutes. As per recommended by AHA, the sphygmomanometer bladder size should be appropriate for the patient. The length of inflating bladder should be at least 80% of the upper arm circumference and the width should be at least 40% of the upper arm circumference. The automated NIBP measurements in monitors follows the oscillometric method. The inflated cuff causes pulsatile pressure changes in the artery, which

produce similar changes in the cuff pressure and this is electronically processed to derive values.

Direct measurement of BP is usually indicated in hemodynamically unstable patients, patients with evidence of end-organ damage or when surgery is associated with major blood loss or fluid shifts. It is invasive and adds much more information from its waveform such as fluid volume status and compliance of arteries.

Q. What are early markers of hypertensive CVD?⁵

Ans. Blood pressure:

- Loss of nocturnal BP dipping
- Exaggerated BP responses to exercise
- Salt sensitivity
- Widened pulse pressure.

Cardiac:

- Left ventricular hypertrophy (mild)
- Increased atrial filling pressure
- Decreased diastolic relaxation.

Vascular:

- Increased central arterial stiffness or pulse wave velocity
- Small artery stiffness
- Increased systemic vascular resistance (SVR)
- Increased wave reflection and systolic pressure augmentation
- Increased carotid intima-media thickness
- Coronary calcification
- Endothelial dysfunction.

Renal:

- Microalbuminuria (urinary albumin excretion of 30–300 mg/dL)
- Elevated serum creatinine
- Reduced estimated glomerular filtration rate (GFR) (60–90 mL/min).

Retinal: Hypertensive retinal changes.

Q. How would you evaluate hypertensive patient for surgery?

Ans. In preoperative evaluation, pay special attention to cause and severity of hypertension, current therapy, and evidence of end-organ damage. When hypertension is detected during preoperative evaluation, by measurement or history, two other issues other than BP control are important—screening for secondary hypertension and extent of target organ dysfunction related to long-standing hypertension. Although secondary hypertension is uncommon, some forms can have serious implications on perioperative management, e.g., pheochromocytoma which can lead to myocardial infarction, heart failure or stroke, or Cushing's syndrome can interfere with postoperative wound healing. Hypertension is also a risk factor for coronary artery disease and cardiomyopathy. When assessing a patient for anesthesia, ask about related illnesses such as ischemic heart disease, renal failure, and

cerebrovascular disease. Those patients with isolated “white coat” hypertension have not been shown to be at higher risk from anesthesia than controls and therefore surgery should not be delayed unnecessarily. Elderly patients with SBPs <180/90 mm Hg should also be considered for surgery, particularly if there is little evidence of end-organ damage, as these values are considered in the normal range for elderly patients due to normal physiological change.

Q. Which are the target organs for hypertension and what are the signs of target organ damage?

Ans. Cardiac:

- Left ventricular hypertrophy (in moderate-to-severe hypertension)
- Systolic or diastolic cardiac dysfunction
- Symptomatic heart failure
- Ischemic heart disease—angina pectoris and myocardial infarction.

Vascular:

- Peripheral arterial disease
- Carotid arterial disease
- Aortic aneurysm
- Wide pulse pressure (>65 mm Hg).

Renal:

- Albuminuria (urinary albumin excretion >300 mg/dL)
- CKD (estimated GFR <60 mL/min) or end-stage renal disease (ESRD).

Cerebrovascular:

- Stroke
- Transient ischemic attack
- Decreased cognitive function
- Dementia
- Loss of vision.

Q. Why is hypertension important?

Ans.

- It is common and incidence in our country is increasing with changing lifestyle.
- It is a risk factor for CVD.
- It causes end-organ damage which affects perioperative course—heart, brain, and kidneys.
- It may indicate the presence of serious endocrine-related diseases—diabetes, thyrotoxicosis, pheochromocytoma, Cushing’s syndrome, Conn’s syndrome, etc. (secondary hypertension).
- It may indicate the presence of serious renal disease (secondary hypertension).
- It increases the risk of an adverse anesthetic outcome/prolonged recovery room stay.
- *Perioperatively, it may be due to:*
 - Anxiety
 - Inadequate anesthesia (awareness)
 - Inadequate analgesia (pain).

Q. What are causes for secondary hypertension?

Ans.

- CKD
- Coarctation of the aorta
- Cushing’s syndrome and other glucocorticoid excess states including chronic steroid therapy
- Drug-induced or drug-related
- Pheochromocytoma
- Primary aldosteronism and other mineralocorticoid excess states
- Renovascular hypertension
- Sleep apnea
- Thyroid or parathyroid disease.

Q. What are screening tests to detect secondary hypertension?

Ans.

- CKD—measuring GFR
- Coarctation of the aorta—echocardiography, magnetic resonance (MR) angiography or CT (computed tomography) angiography
- Cushing’s syndrome and other glucocorticoid excess states including chronic steroid therapy—history and dexamethasone suppression test
- Drug-induced/related—history and drug screening
- Pheochromocytoma—24-hour urinary metanephrine and normetanephrine
- Primary aldosteronism and other mineralocorticoid excess states—24-hour urinary aldosterone level or specific measurements of other mineralocorticoids
- Renovascular hypertension—Doppler flow study or MR angiography of renal vessels
- Sleep apnea—sleep study (polysomnography)
- Thyroid/parathyroid disease—TSH (thyroid-stimulating hormone) and serum PTH (parathyroid hormone).

Q. Which drugs can cause hypertension?

Ans.

- Sympathomimetic drugs such as amphetamine, cocaine, and migraine medications
- Steroids
- Immunosuppressants such as cyclosporine and tacrolimus
- Estrogen and oral contraceptive pills
- Serotonin-norepinephrine reuptake inhibitors
- Caffeine.

Q. What is white coat hypertension?

Ans. White coat effect (WCE), or white coat phenomenon, is the transient rise in BP from before to during the visit to the doctor’s clinic, which settles down after a period of rest. White coat hypertension is due to increased anxiety leading to high BP recorded in a medical setting with normal ambulatory BP response.

Q. Explain pathophysiology of essential hypertension.

Ans. Blood pressure is typically defined as lateral pressure exerted by blood on vessel wall mainly in arteries. Since mean arterial pressure (MAP) is the product of cardiac output (CO) and SVR, hypertension can result due to either increase in CO or increased SVR. Of all hypertensive patients, 90% have essential or primary hypertension where underlying pathophysiology is increased SVR. About 70–80% patients with primary hypertension have family history of hypertension suggesting strong genetic influence. Since the autonomic nervous system (ANS) plays a central role in the maintenance of cardiovascular homeostasis, disturbance of ANS, such as sympathetic nervous system overactivity, increases BP and contributes to the development and maintenance of hypertension. The increased sympathetic nervous system activity in hypertension is multifactorial and complex and involves alterations in baroreflex and chemoreflex pathways at both peripheral and central levels. Arterial baroreceptors as well as aortic baroreceptors are reset to a higher pressure in hypertensive patients. Angiotensin II, endothelin, and reactive oxygen radicals have been found to play major role in resetting baroreceptors.

Peripheral vasculature: The arterial and arteriolar walls are thickened with resultant high ratio of wall thickness to internal diameter. There is relative hypovolemia due to which vascular relaxation following induction of anesthesia leads to a greater than expected decrease in BP. Similarly, vascular contraction leads to an abnormally large increase in BP.

Heart: In order to cope with the increased afterload, the left ventricular myocardium hypertrophies. This leads to increased wall tension, increased myocardial O₂ requirement, and endomyocardial fibrosis. Cardiac dilatation eventually follows, and subsequently heart failure.

Electrocardiogram (ECG) shows left ventricular hypertrophy with strain. Myocardial compliance is decreased with resultant increase in the atrial contribution to diastolic filling and CO. Patients may develop congestive cardiac failure and pulmonary edema. The risk for perioperative myocardial ischemia is increased in hypertensive patients with left ventricular hypertrophy.

Brain: Chronic hypertension shifts cerebral and renal autoregulation to the right, requiring maintenance of higher BP to maintain cerebral blood flow as compared to normotensive patients. With long-term therapy, autoregulation curve shifts to normal. Recent study conducted by Zhang et al. demonstrated that in patients with mild and moderate hypertension autoregulation

returns to normal within 1–2 weeks of treatment.⁶ However, in severely hypertensive patients, this period may be longer. Remember that hypertensive patients may also have cerebrovascular and carotid arterial disease and in “Watershed” area, ischemia may occur with even relative hypotension.

Kidneys: End-organ damage to the kidneys from hypertension can lead to glomerular sclerosis, abnormal distribution of renal blood flow, and decreased GFR. Hypoperfusion due to a sudden and sustained decrease in BP (during anesthesia for major surgery) can lead to postoperative renal insufficiency.

Q. Discuss relevance of commonly used antihypertensive drugs to an anesthetist. How will you control hypertension in individual patients?

Ans. Described in Table 2.

TABLE 2: Relevance of commonly used antihypertensive drugs.

Mechanism of action	Examples	Relevance to anesthesia
Diuretics	Thiazide, frusemide, spironolactone, etc.	Electrolyte disturbance
Calcium channel blockers	Nifedipine and verapamil	May cause precipitous hypotension
ACE inhibitors	Captopril, enalapril, ramipril, etc.	Can lead to severe catecholamine resistant hypotension under anesthesia
β-blockers	Propranolol, metoprolol, atenolol, and labetalol	Avoid in asthmatics and patients with overt congestive heart failure (CHF), peripheral vascular disease, and bradyarrhythmias. Failure to mount tachycardia in instances of major blood/fluid loss
Angiotensin receptor blockers	Losartan, valsartan, and telmisartan	Same as ACE inhibitors
Vasodilators	Hydralazine and diazoxide	Tachycardia
Centrally acting	Clonidine, methyl dopa, and reserpine	Rebound hypertension, if withdrawn
Alpha blockers (not used recently)	Phenoxybenzamine and phentolamine	Tachycardia

(ACE: angiotensin-converting enzyme).

Therapy should begin with lifestyle modification, and if BP goal is not achieved, antihypertensive drugs should be started using one of the first-line agents (thiazides,

CCBs, ACE inhibitors, and ARBs) that have been shown to reduce one or more hypertensive complications in randomized controlled trials. The choice of drugs also depends on the presence of comorbidity. For example, in patients with coronary artery disease, β -blocker would be the drug of choice, whereas in patients with diabetes or renal dysfunction, ACE inhibitor would be the drug of choice. If the initial drug selected is not tolerated or is contraindicated, then a drug from one of the other classes proven to reduce cardiovascular events should be substituted. Since most hypertensive patients will require two or more antihypertensive medications to achieve their BP goals, addition of a second drug from a different class should be initiated when use of a single agent in adequate doses fails to achieve the goal. Use of more than one drug increases the likelihood of achieving BP goal. The use of multidrug combinations helps in better BP control at lower doses of the component agents, with fewer side effects. The ACC/AHA 2017 guidelines for hypertension recommend initiation of antihypertensive treatment with two first-line drugs from different classes in patients with stage 2 hypertension.³

Q. What is the BP response to perioperative procedure in a hypertensive patient?

Ans. In a truly hypertensive patient, BP response can be considered in two phases—during anesthesia and postoperative period. Generally, these patients display fall in BP during induction, followed by pressor response and tachycardia during laryngoscopy and intubation, a stabilization of BP and heart rate during anesthesia and another rise in BP and heart rate during reversal and extubation. Sympathetic activation during laryngoscopy and intubation do cause rise in BP and the heart rate in normotensive individuals. However, these responses are more pronounced in patients with untreated and inadequately treated hypertensive individuals. These excessive swings in BP and heart rate can increase risk of ischemia or cardiac failure for patients with coronary artery disease (CAD) or left ventricular dysfunction. Hypertensive patients are known to have increased incidence of ST segment depression during surgery. In addition, chronic uncontrolled or poorly controlled hypertension resets cerebral autoregulation at a higher level as mentioned above, so that in these patients, even moderate fall in BP in perioperative period may lead to cerebral ischemia.

Q. How would you anesthetize this patient?

Ans.

Premedication: Patient should receive good premedication in the form of anxiolytics. Premedication with benzodiazepines (e.g., diazepam 5–10 mg or alprazolam 0.25–0.5 mg) on night

before and 2 hours prior to surgery will help in allaying anxiety and associated rise in BP. Premedication with α_2 -adrenoceptor agonists may be useful. Clonidine provides hemodynamic stability and reduces the risk of myocardial ischemia by reducing sympathoadrenal activity. In addition, clonidine also causes anxiolysis, sedation, and decreases the need for both inhalation and intravenous (IV) anesthetics. Atropine should be avoided if possible, because of its tendency to cause tachycardia. Anesthetic management should be directed toward avoidance of both hypotension and hypertension.

Induction: If the patient shows significant hypertension in the preinduction area, a small dose of intravenous sedation (midazolam 1–3 mg) may help to control it. Care should be taken not to cause a precipitous fall in arterial pressure at induction. The use of opioids, such as morphine or fentanyl, in liberal doses will attenuate response to laryngoscopy and intubation and will also reduce the amount of induction agent required. Etomidate is most cardiostable, but thiopentone may be used provided it is given slowly, and titrated against response. Propofol may cause precipitous hypotension and ketamine, which raises the arterial pressure and heart rate and hence should be avoided. Perform smooth, gentle, and fast (<30 seconds) laryngoscopy and intubation to minimize sympathetic response. Spraying of vocal cords is found to be ineffective in preventing this response and no longer recommended. Use of IV lignocaine in the dose of 1.5 mg/kg has been widely practiced for the same purpose, but various studies have shown it to be ineffective or less effective as compared to drugs such as β -blockers, vasodilators, CCBs, and high-dose opioids. Atracurium, rocuronium, and vecuronium are suitable muscle relaxants. Severe hypotension after induction may be more harmful than hypertension and hence one should keep vasopressors ready and treat hypotension promptly. All patients should have continuous monitoring of the ECG and oxygen saturation. Intraoperative BP monitoring may be noninvasive or invasive depending on type of surgery, expected fluid shifts, and presence of comorbidity.

Maintenance: High concentrations of volatile agents can cause hypotension by decreasing the SVR and by depressing the myocardium. The liberal use of opiates, which have minimal cardiovascular effects, will reduce the amount of volatile agents required. ENIGMA (Evaluation of Nitrous Oxide in the Gas Mixture for Anesthesia) I trial published in 2011 had showed a nonsignificant increase (from 0.7 to 1.3%, $p = 0.26$) in ischemic cardiac complications within 30 days of surgery with the use of nitrous oxide; however, ENIGMA II published in 2014 failed to confirm these results and thus concluded that nitrous oxide can be safely used without increased cardiac risk for patients

undergoing major noncardiac surgeries.^{7,8} Local anesthetic nerve blocks or infiltration are useful either on their own or in addition to general anesthesia. Hypertension during anesthesia may reflect inadequate depth of anesthesia, hypoxia, or hypercarbia due to inadequate ventilation. These factors should be corrected before treating PB with antihypertensive drugs. Avoid perioperative hypothermia. Hypotension should be vigorously treated by reducing the concentration of volatile anesthetic agent (not too low otherwise patient might develop awareness) and correcting any hypovolemia as hypertensives tolerate hypovolemia poorly. The reduced left ventricular compliance and more rigid vascular tree found in hypertensive patients make them more vulnerable to small changes in blood volume. Furthermore, β -blockers prevent the physiological heart rate changes, whereas vasodilators prevent vasoconstrictive response. Thus, careful monitoring of blood and fluid losses is important and prompt replacement of fluid deficit is necessary to avoid undue hypotension. Bradycardia should be treated using titrated doses of IV atropine. Patients on β -blockers are particularly liable to develop bradycardia, and may develop marked cardiac depression with anesthetic agents. Occasionally, a small dose of a vasopressor may be required in patients not responding to this approach. Normal intravenous fluid replacement should be given.

Recovery: Coughing on the tracheal tube during emergence will increase arterial pressure. Opioids given during anesthesia reduce this tendency. These patients should be extubated in deeper planes if airway is safe.

Role of regional anesthesia: Spinal and epidural anesthesia can cause unpredictable and profound arterial hypotension in poorly controlled hypertensive patients. Well-controlled hypertensives, however, have a more predictable response. Peripheral nerve blocks, e.g., brachial plexus blocks or ankle blocks, should always be considered in hypertensive patients, as the potential hazards of general anesthesia are avoided.

During emergency surgery, aim for intraoperative BP around 140/90 mm Hg but avoid swings $>\pm 20\%$ from baseline BP. Urine output should be closely monitored.

Q. Should we treat isolated systolic hypertension?

Ans. There is a steady increase in systolic arterial pressure starting in childhood and continuing throughout adult life. In contrast, diastolic pressure rises in early adult life and then stabilizes or declines in the fifth and sixth decades of life. Isolated systolic hypertension is the situation in which the diastolic arterial pressure is normal, but the systolic arterial pressure is elevated. Franklin et al.⁹ found that 80% of the subjects aged over 50 years who

had hypertension had isolated systolic hypertension. Isolated systolic hypertension is associated with a small but statistically significant increase in the likelihood of perioperative morbidity and benefits of treating isolated systolic hypertension are clearly established as major cardiovascular events are reduced.

Q. What are the causes of intraoperative hypertension? How would you manage it?

Ans. Hypertension occurs commonly during anesthesia. Main reasons are—drug errors (inadvertent injection of vasopressors), infiltration with vasopressors, awareness or light plane of anesthesia, preexisting hypertension, airway problems (malfunctioning valves and hypoventilation), endobronchial intubation, surgical factors (direct sympathetic stimulation), hypercarbia, uncommon conditions (pheochromocytoma, hyperthyroidism, and malignant hyperthermia), raised intracranial pressure, and fluid overload.

Management: First confirm that BP change is real (check BP cuff/transducer level in case of invasive BP), check for ventilation malfunction, ensure adequate alveolar minute ventilation, give adequate analgesia, deepen plane of anesthesia, inform surgeons and interrupt surgery if necessary, and recheck the drug errors. Only after these steps have been taken consider using antihypertensive drugs. One can use nitroglycerin (NTG) 20 mg diluted in 20–50 mL 5% dextrose or normal saline through syringe pump and titrate as per response. If tachycardia is troublesome use β -blockers such as metoprolol. Intravenous infusion of labetalol at a rate of 0.5 mg/kg/h and titrated to response is better option. Labetalol being α - and β -blocker does not give rise to tachycardia as seen with NTG or sodium nitroprusside.

Q. What are the perioperative issues in patients on antihypertensive drugs?

Ans. ACE inhibitors block angiotensin-converting enzyme. ACE inhibitors and adrenergic receptor blockers (ARBs) will usually be continued prior to anesthesia, but one may consider omitting these drugs in preoperative period if major blood loss is anticipated or regional techniques such as epidurals are carried out, as the cumulative hypotensive effects may be detrimental. Renal function should be carefully monitored in those patients taking ACE inhibitors, particularly in the perioperative period. Nonsteroidal anti-inflammatory drugs should be used with caution in patients taking ACE inhibitors. Some authors recommend stopping ACE inhibitors and ARBs and substituting other antihypertensive agent 1 day prior to surgery to prevent precipitous fall in BP especially for surgeries where massive blood loss or major fluid shifts are expected.

β -blockade has been previously shown to improve perioperative morbidity and mortality in patients with ischemic heart disease, although a more recent perioperative ischemic evaluation (POISE) study showed increased central nervous system morbidity and mortality if β -blocker drug is started in the preoperative period; however, if patient is already on β -blocker, it should be continued in the preoperative period. β -blockers are better avoided in patients with reactive airway disease. Also, particular caution should be exercised in patients on β -blockers as these drugs mask manifestation of tachycardia which may be due to pain, reduced CO, awareness, anaphylaxis, or even malignant hypertension during anesthesia.

Q. Enumerate problems of anesthetizing a patient with uncontrolled hypertension.

Ans.

- Cardiac ischemic events (especially in the presence of left ventricular hypertrophy)
- Arrhythmias
- Cerebral autoregulation reset (shift to right) making patients prone for cerebrovascular accidents (CVAs)
- Exaggerated cardiovascular response to intubation and extubation
- Presence of diastolic dysfunction (poor left ventricular relaxation) makes patients prone to pulmonary edema
- Excessive surgical bleeding.

Q. A 52-year-old man presents to surgery for inguinal hernia repair. In preoperative check, you find his BP 200/120 mm Hg. What would you do?

Ans. Recheck the BP after assuring the patient and adequate period of rest to rule out WCE. Check that BP cuff size is adequate for the patient. Ask a detailed history whether he has preexisting hypertension and on any medication.

Q. On rechecking, his BP is same and patient does not give history of hypertension, though complains of headaches, decreased effort tolerance, and easy fatigability since 6–8 months.

Ans. We should not go ahead with the planned surgery and will have to wait till his BP is under control. Explain the patient about need to control BP and implications of anesthesia risk and long-term risk to life in case of uncontrolled hypertension. The patient should undergo investigations to rule out presence of secondary hypertension. Start treatment with any of the drugs given in **Table 2** and refer to physician for proper evaluation and treatment.

The 2014 ACC/AHA guideline developed for perioperative cardiovascular evaluation in patients undergoing noncardiac surgery states that uncontrolled systemic hypertension is a minor clinical predictor of increased perioperative cardiovascular risk. If the initial

evaluation establishes hypertension as mild or moderate and there are no associated metabolic or cardiovascular abnormalities, there is no evidence suggesting beneficial effects of delaying surgery. However, for severe hypertension (>180/>110 mm Hg), the potential benefits of delaying surgery to optimize the effects of antihypertensive medications should be weighed against the risk of delaying the surgical procedure. Control can be achieved over several days to weeks of preoperative outpatient treatment. The guideline emphasizes that continuation of preoperative antihypertensive treatment throughout the perioperative period is critical.¹⁰

According to Miller, elective surgery should be delayed for severe hypertension, i.e., DBP >115 mm Hg and SBP >200 mm Hg until BP is <180/110 mm Hg. Preoperative BP charting of the patient also helps in decision. If the BP is 180/100 mm Hg with no signs of myocardial ischemia, the patient is likely to tolerate the same intraoperatively as well. Similarly, if patient experiences low BP in the wards, without cerebral deficit, he/she is likely to tolerate the same in the perioperative period. Hence, individualized thresholds can be ascertained. All antihypertensives are to be continued preoperatively except ACE inhibitors and ARBs due to increased associated risk of intraoperative hypotension.

Q. How soon the patient can undergo surgery?

Ans. There are no definite recommendations about how long you need to have patient's numbers normalized before subjecting them to anesthesia. A study by Zhang showed that in patients with mild-to-moderate hypertension autoregulation returns to normal within 1–2 weeks of treatment. Thus, for this category of patients, it would be safe to anesthetize after 1–2 weeks of normalization of BP. However, for patients with severe hypertension, there are no studies looking at normalization of BP and return of autoregulation limits to normal. One has to keep in mind that the numbers may normalize quickly but several weeks of treatment may be necessary to reduce abnormal vascular tone and autoregulatory threshold to get resettled.

Q. You are called to recovery to review a 77 years old patient who has undergone laparoscopic cholecystectomy, his BP is 210/110 mm Hg. How will you manage this patient?

Ans. The common cause of postoperative hypertension are:

- Pain
- Emergence phenomenon
- Hypercarbia
- Intolerance of endotracheal tube
- Full bladder
- Hypervolemia
- Peripheral vasoconstriction due to hypothermia and hypovolemia
- Withdrawal of chronic therapy.

Postoperative hypertension can lead to:

- Damage to vascular anastomoses
- Postoperative bleeding
- Intracranial bleeding
- Myocardial ischemia (particularly when associated with tachycardia).

Treatment of postoperative hypertension depends on the clinical situation, the etiology, and the degree of hypertension. Analgesia must be optimized. Hypertension, when severe, should be treated with NTG, labetalol, esmolol, nitroprusside, hydralazine, etc.; sublingual nifedipine should be avoided as it can lead to uncontrolled and precipitous fall in BP.

This patient has severe hypertension. Rule out above-mentioned causes. Repeat BP measurements regularly in addition to other routine monitoring. If hypertension persists, treat with labetalol or NTG.

Q. What is malignant hypertension or hypertensive crisis? How will you manage it?

Ans. Malignant hypertension is an older terminology and no longer used. Hypertensive crisis is a more valid term and includes two different diagnoses, hypertensive emergency (HE) and hypertensive urgency (HU). JNC-7 defines HE as the "clinical situations characterized by severe elevations in BP ($>180/120$ mm Hg), complicated by evidence of impending or progressive target organ dysfunction." JNC-7 defines HU as "those situations associated with severe elevations in BP without progressive target organ dysfunction." There is no absolute value of BP that separates HE from HU. Instead, the most important distinction is whether there is evidence of impending or progressive end-organ damage, which defines an emergency. The common causes of hypertensive crises are as follows:

- Antihypertensive drug withdrawal (e.g., clonidine)
- Autonomic hyperactivity
- Drugs such as cocaine and amphetamines
- Acute glomerulonephritis
- Head trauma
- Pheochromocytoma
- Preeclampsia and eclampsia
- Renovascular hypertension.

Pathophysiology

- Abrupt increases in SVR likely due to humoral vasoconstrictors
- Failure of autoregulatory mechanism in vascular bed leading to increased mechanical stress and endothelial injury
- Activation of renin-angiotensin system
- Platelet activation and development of prothrombotic state
- Ischemia causing release of vasoactive substances leading to a vicious cycle.

Clinical manifestations: The manifestations of hypertensive emergencies are those of end-organ dysfunction:

- Hypertensive encephalopathy
- Acute aortic dissection
- Acute myocardial infarction
- Intracerebral bleed
- Eclampsia
- Unstable angina
- Acute left ventricular failure with pulmonary edema.

Hypertensives urgencies are upper levels of stage II hypertension associated with:

- Severe headache
- Shortness of breath
- Epistaxis
- Severe anxiety.

It is important to recognize that the absolute value of BP may not be as important as the rate of increase in BP. Patients with long-standing hypertension may tolerate SBP of 200 mm Hg or DBP of up to 150 mm Hg without developing hypertensive encephalopathy, while children or pregnant women may develop encephalopathy with DBP of 100 mm Hg.

Hypertensive Encephalopathy

- Headache
- Altered level of consciousness and confusion
- Acute CVA with intraventricular bleeding or ischemic infarction resulting in focal neurological abnormalities
- Fundoscopy showing advanced retinopathy with arteriolar changes, hemorrhages, exudates, papilledema.

Cardiovascular Manifestations

- Angina
- Left ventricular failure
- Acute myocardial infarction
- *Aortic dissection:* Propagation of the dissection is dependent not only on the elevation of the BP itself, but also on the velocity of left ventricular ejection.

Renal Manifestations

Renal failure with oliguria and/or hematuria.

Treatment

Intravenous sodium nitroprusside, labetalol, esmolol, and NTG can be used for the treatment of malignant hypertension. BP should be reduced by 10–15% (maximum of 20%) in a controlled fashion within the first hour with a continued decrease toward 160/100 mm Hg over the next 2–6 hours as tolerated by the patient. A more rapid reduction is indicated in patients with aortic dissection. Doses, duration of action, and adverse effects of drugs used for hypertensive emergencies are given in **Table 3**.

TABLE 3: Drugs for treatment of hypertensive emergencies.

Drug	Dose	Onset of action	Duration of action	Adverse effects	Special indications
<i>Vasodilators</i>					
Sodium nitroprusside	0.25–10 µg/kg/min as IV infusion	Immediate	1–2 minutes	Nausea, vomiting, muscle twitching, sweating, thiocyanate, and cyanide intoxication	Most hypertensive emergencies; caution with high intracranial pressure or azotemia
Nitroglycerin	5–100 µg/min as IV infusion	2–5 minutes	5–10 minutes	Headache, vomiting, methemoglobinemia, and tolerance with prolonged use	Coronary ischemia
Fenoldopam	0.1–0.3 µg/kg/min IV infusion	<5 minutes	30 minutes	Tachycardia, headache, nausea, and flushing	Most hypertensive emergencies; caution with glaucoma
Nicardipine hydrochloride	5–15 mg/h IV	5–10 minutes	15–30 minutes, may exceed 4 hours	Tachycardia, headache, flushing, and local phlebitis	Most hypertensive emergencies except acute heart failure; caution with coronary ischemia
Hydralazine hydrochloride	10–20 mg IV 10–40 mg IM	10–20 minutes IV 20–30 minutes IM	1–4 hours IV 4–6 hours IM	Tachycardia, flushing, headache, vomiting, and aggravation of angina	Eclampsia
<i>Adrenergic inhibitors</i>					
Labetalol hydrochloride	20–80 mg IV bolus every 10 minutes 0.5–2.0 mg/min IV infusion	5–10 minutes	3–6 hours	Vomiting, scalp tingling, bronchoconstriction, dizziness, nausea, heart block, and orthostatic hypotension	Most hypertensive emergencies except acute heart failure
Esmolol hydrochloride	250–500 µg/kg/min IV bolus, then 50–100 µg/kg/min by infusion; may repeat bolus after 5 minutes or increase infusion to 300 µg/min	1–2 minutes	10–30 minutes	Hypotension, nausea, asthma, first-degree heart block, and heart failure	Aortic dissection and perioperative
Phentolamine	5–15 mg IV bolus	1–2 minutes	10–30 minutes	Tachycardia, flushing, and headache	Catecholamine excess as in pheochromocytoma

Q. How would you manage patients with uncontrolled hypertension for surgery?

Ans. Uncontrolled hypertension is associated with wider fluctuations of BP during induction of anesthesia and intubation, and may increase the risk for perioperative ischemic events. BP levels of >180/110 mm Hg should be controlled prior to surgery. In urgent situations, rapidly acting parenteral agents, such as sodium nitroprusside and labetalol, can be utilized to achieve effective control very rapidly. Care should be taken not to cause precipitous fall. Most of the times these patients would have occult hypovolemia and would need proper fluid management. Continuous beat-to-beat monitoring of BP is advisable

while using these drugs. Candidates with controlled hypertension should maintain their medications until the time of surgery, and therapy should be reinstated as soon as possible postoperatively. Since most hypertensive patients are on diuretic drugs, hypokalemia and hypomagnesemia may be present and should be corrected prior to anesthesia. Sudden intraoperative hypertension is managed by same antihypertensive agents that are utilized in the management of hypertensive emergencies. NTG is often an agent of choice in patients with coronary ischemia, while the very short-acting β -blockers, such as esmolol, may be of benefit in managing intraoperative tachycardia and hypertension. Hypertension is very common in the

early postoperative period and is related to increased sympathetic tone and vascular resistance. Contributing factors include pain and increased intravascular volume, which may require parenteral dosing with a loop diuretic such as furosemide. If resumption of oral treatment must be interrupted postoperatively, periodic dosing with intravenous antihypertensive agents should be considered.

■ REFERENCES

1. James PA, Oparil S, Carter BL, Cushman WC, Dennison-Himmelfarb C, Handler J, et al. 2014 evidence-based guideline for the management of high blood pressure in adult report from the panel members appointed to the Eighth Joint National Committee (JNC 8). *JAMA*. 2014;311(5):507-20.
2. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, et al. Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Hypertension*. 2003;42(6):1206-52.
3. Whelton PK, Carey RM, Aronow WS, Casey DE Jr, Collins KJ, Dennison Himmelfarb C, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/ NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: Executive Summary: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Hypertension*. 2018;71(6):1269-324.
4. Williams B, Mancia G, Spiering W, Agabiti Rosei E, Azizi M, Burnier M, et al. 2018 ESC/ESH Guidelines for the management of arterial hypertension. *Eur Heart J*. 2018;39(33):3021-104.
5. Giles TD, Materson BJ, Cohn JN, Kostis JB. Definition and classification of hypertension: an update. *J Clin Hypertens*. 2009;11(11):611-4.
6. Zhang R, Witkowski S, Qi Fu, Claassen JA, Levine BD. Cerebral hemodynamics after short- and long-term reduction in blood pressure in mild and moderate hypertension. *Hypertension*. 2007;49(5):1149-55.
7. Leslie K, Myles PS, Chan MT, Forbes A, Paech MJ, Peyton P, et al. Nitrous oxide and long-term morbidity and mortality in the ENIGMA trial. *Anesth Analg*. 2011;112(2):387-93.
8. Myles PS, Leslie K, Chan MT, Forbes A, Peyton PJ, Paech MJ, et al. The safety of addition of nitrous oxide to general anaesthesia in at-risk patients having major non-cardiac surgery (ENIGMA-II): a randomised, single-blind trial. The ANZCA Trials Group for the ENIGMA-II investigators. *Lancet*. 2014;384(9952):1446-54.
9. Franklin SS, Gustin W 4th, Wong ND, Larson MG, Weber MA, Kannel WB, et al. Hemodynamic patterns of age-related changes in blood pressure. The Framingham Heart Study. *Circulation*. 1997;96(1):308-15.
10. Fleisher LA, Fleischmann KE, Auerbach AD, Barnason SA, Beckman JA, Bozkurt B, et al. 2014 ACC/AHA guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery. A report of the American College of Cardiology/American Heart Association Task Force on practice guidelines. *J Am Coll Cardiol*. 2014;64(22):e77-137.

Tetralogy of Fallot

Vandana Agarwal, Reshma Ambulkar, Madhavi Desai

An 8-month-old boy is admitted with history of mild cyanosis since 2 weeks after birth and gradually increasing over the last 3 months and worsening on crying. He was diagnosed to have tetralogy of Fallot on cardiac catheterization.

Q. How is a pediatric cardiac surgery patient different from an adult cardiac surgery patient?

Ans. The cardiovascular system in a neonate and infant has reduced myocardial reserve. This is due to the following reasons:

- Limited or restricted number of β -receptors
- High resting levels of circulating catecholamines
- An immature calcium transport system. In contrast to adult myocardium, they depend on the extracellular calcium levels
- Reduced ventricular compliance
- The stroke volume does not increase with preload augmentation. As a result, they are more dependent on the heart rate to increase the cardiac output.

Pediatric patients are physically smaller than adults. To establish invasive monitoring is challenging in these small patients. Sometimes it may not be possible to use conventional monitoring such as pulmonary artery catheter (in patients <7 kg, 5.0 Fr pulmonary artery catheter may have to be inserted through the femoral vein rather than internal jugular vein and may require fluoroscopic guidance). One may have to rely on transthoracic catheters inserted during the time of surgery as well as specialized monitoring such as transesophageal or epicardial echocardiography with Doppler color flow imaging.

During cardiopulmonary bypass (CPB), these patients are cooled to 15–18°C; hemodilution occurs to >50% of their extracellular volume. They may undergo periods (>1 hour) of deep hypothermic cardiac arrest. They have exaggerated inflammatory response to CPB due to the disproportionate exposure to the nonendothelialized surfaces of the CPB circuit per body surface area compared to adults.

Q. What is tetralogy of Fallot (ToF)?

Ans. Tetralogy of Fallot is a tetrad of anatomical malformations resulting from a single morphologic defect, i.e., malalignment of the anterior part of the infundibular septum. The four components are:

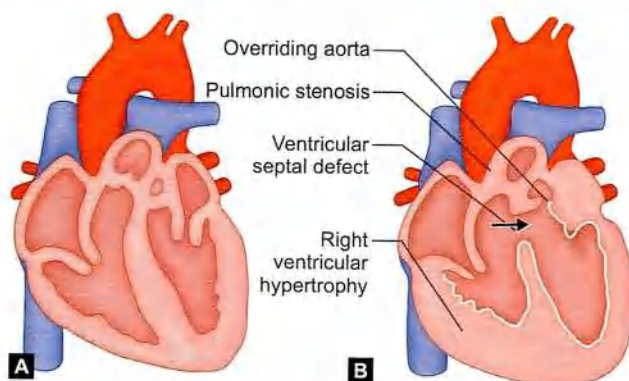
1. Right ventricular outflow tract obstruction (RVOTO)
2. Ventricular septal defect (VSD)
3. An overriding aorta
4. Right ventricular (RV) hypertrophy.

The RVOTO may be infundibular spasm, pulmonary stenosis, or pulmonary atresia. ToF with pulmonary atresia is a much more severe form of congenital heart disease (CHD) and cyanosis develops almost immediately after birth and worsens progressively as the ductus arteriosus closes. These patients depend on the patent ductus arteriosus until definitive corrective surgery is done. Prostaglandins are added to maintain the patency of ductus arteriosus. ToF with pulmonary atresia may be associated with other syndromes such as acronyms:

- CATCH22 (cardiac defect, abnormal face, thymic hypoplasia, cleft palate, hypocalcemia, associated with microdeletion of band 22q11)
- VATER syndrome includes the vertebral defects, anal atresia, tracheoesophageal fistula with esophageal atresia, and renal and radial anomalies.
- CHARGE syndrome includes the coloboma, heart disease, choanal atresia, retarded growth and retarded development and/or central nervous system (CNS) anomalies, genital hypoplasia, and ear anomalies and/or deafness (**Figs. 1A and B**).

The diagnosis can be done prenatally with fetal ultrasound. In acyanotic patients, a crescendo-decrescendo systolic murmur—best heard at the upper left sternal border—is audible at birth. They may be asymptomatic

and later on develop signs and symptoms of congestive heart failure (CHF) secondary to large left to right shunt. Most patients become cyanotic after birth. Subsequently, echocardiography can be done to confirm the diagnosis.



Figs. 1A and B: (A) Normal heart; (B) Tetralogy of Fallot.

The nature of the right ventricular outflow tract (RVOT) dictates the onset and severity of symptoms. If untreated, the infant progressively becomes cyanotic because of worsening infundibular stenosis and develops polycythemia in an attempt to increase oxygen delivery to the tissues. Hypoxic spells may occur in infants with agitation and crying. If cyanosis is severe, it can lead to hypotension, hemodynamic instability, and finally loss of consciousness. Persistent cyanosis may cause growth retardation. Polycythemia predisposes to brain abscess and stroke.

Tetralogy of Fallot is the most common cyanotic heart defect seen in children; if treated, it results in survival to adulthood. Also, it is the most common complex lesion encountered after repair in adult population. If untreated, 30% of the infants with ToF and pulmonary stenosis die within the first year, 40% by 4 years, 70% by 10 years, and 95% by 40 years. In postsurgical repair, these patients can get sudden cardiac arrest secondary to atrial reentrant tachycardia and/or high-grade ventricular arrhythmias. These patients may be posted for insertion of implantable cardioverter device.

Q. What is the pathophysiology of ToF?

Ans. There are two pathophysiologic problems:

1. Reduced pulmonary blood flow results in systemic hypoxemia and cyanosis
2. Increased impedance to RV ejection results in pressure overload, ventricular dysfunction, and failure of the RV.

The VSD leads to an increase in the blood flow and pressure in the right ventricle causing RV hypertrophy. The severity of the shunt increases with the severity of RVOTO. Chronic hypoxia occurs because of shunting of blood.

Exacerbations of hypoxia following infundibular spasm may be life-threatening. Chronic hypoxia leads to polycythemia which predisposes to hypercoagulability and thrombosis. Since right-to-left shunting of blood bypasses the filtering effect of the pulmonary capillaries, these patients have a higher incidence of systemic infection such as a brain abscess.

Q. What are the clinical features?

Ans.

- Cyanosis may be present immediately or shortly after birth and increases progressively thereafter as RV hypertrophy develops increasing the shunt fraction and worsening systemic hypoxemia.
- The neonate or infant may present with cyanosis during feeding, fatigue during feeding, irritability, and tachypnea. Fatigue during feeding and tachypnea decreases the time for swallowing leading to inadequate feed intake and therefore failure to thrive.
- Dyspnea on exertion can occur following crying, feeding in an infant, or while playing or running in an older child.
- *Hypoxic spells:* Parent may mention that the child squats when he/she becomes blue.
- Physical growth retardation.

Q. What are the compensatory mechanisms in patients with ToF?

Ans.

- RV hypertrophies to work against narrow RVOT
- Polycythemia in the face of chronic hypoxia
- Squatting may be considered a compensatory mechanism by the child to attenuate cyanotic spells.

Q. What is a pink tet?

Ans. The term pink tet refers to ToF with minimal valvular or subvalvular stenosis. Blood flow to the pulmonary circulation is sufficient to maintain the deoxygenated hemoglobin concentration <5 g%. As a result, these patients are acyanotic. This term is also applied to patients with ToF with pulmonary atresia or severe pulmonary stenosis with patent ductus arteriosus and those with major aortopulmonary collateral arteries. An acyanotic patient with ToF (pink tet) has a long, loud, crescendo-decrescendo systolic murmur—best heard at the upper left sternal border. The loud murmur is a reflection of mild RVOT. Infants with acyanotic ToF (pink ToF) may be asymptomatic or may show signs of CHF from a large left to right shunt. In acyanotic patients with CHF, medical management is similar to that of a patient with a VSD and includes diuretics (furosemide), digoxin, and afterload reduction with angiotensin-converting enzyme inhibitors (captopril).

Q. What is a hypoxic/hypercyanotic/tet spell?

Ans. "Tet spell" is paroxysmal exacerbation of hypoxemia usually preceded by hyperpnea. The exact etiology is not fully understood; it occurs probably due to infundibular spasm aggravating the RVOTO which leads to worsening of right to left shunt causing acute severe cyanosis and may lead to loss of consciousness. Hypoxemia further increases the right to left shunt by decreasing the systemic vascular resistance (SVR).

It is characterized by paroxysms of hyperpnea, prolonged crying, intense cyanosis, and decreased intensity of the murmur of pulmonic stenosis as the blood is flowing past the VSD from right to left ventricle. It is precipitated by activity or distress (crying, defecating, agitation, or fright) and squatting relieves the spell. Squatting causes compression of the femoral vessels, thus increasing the SVR which reduces shunt from right to left across the VSD and therefore reduces cyanosis. Other factors which can worsen cyanosis are anemia, acidosis, infection, and stress.

"Tet spells" can also be precipitated by an acute reduction in SVR, such as during induction of anesthesia (especially if the patient is hypovolemic). The anesthetist should be prepared to manage a hypoxic spell.

Q. How do you manage a tet spell?

Ans. The main aim of management is to increase the SVR and decrease the PVR:

- *Knee-chest position:* It causes compression of the femoral vessels increasing the SVR and thus decreasing the right to left shunt. Place the baby on the mother's shoulder with the knees tucked up underneath. This also provides a calming effect.
- Oxygen is of limited value since the primary abnormality is reduced pulmonary blood flow and increased right to left shunt. Administer oxygen.
- Morphine sulfate, 0.1–0.2 mg/kg IV, can be given to reduce the respiratory drive and hyperpnea.
- Phenylephrine 5–10 µg/kg IV or as an infusion 2–5 µg/kg/min can be used to increase the SVR.
- Dexmedetomidine infusion, 0.2 µg/kg/min, has recently been shown to ameliorate symptoms during the hypoxic spell.
- Volume resuscitation either with crystalloids or colloids (15–30 mL/kg) augments the RV preload, which may increase the diameter of the heart and thus physically decrease the RVOTO.
- β-blockers can be administered to decrease infundibular spasm. They have a myocardial depressant effect and thus relieve the infundibular spasm. Also, decrease in the heart rate improves the diastolic filling of the ventricle and thus decreases RVOTO. Esmolol 0.5 mg/kg

bolus IV followed by an infusion of 50–300 µg/kg/min or propranolol 0.1 mg/kg can be used.

- Give 100% O₂.

Q. What surgical procedures are available to correct this condition?

Ans.

Palliative Surgery

The principle is to increase the pulmonary blood flow by creating a shunt between systemic and pulmonary circulation. Shunt surgery improves oxygenation and promotes pulmonary vascular growth but predisposes to RV failure secondary to increased pulmonary blood flow (Fig. 2). Various procedures have been described which are as follows:

- Blalock-Taussig shunt (BT shunt) is a direct end-to-end anastomosis of subclavian artery to pulmonary artery.
- Potts procedure is the anastomosis of descending aorta to pulmonary artery.
- Waterston shunt is the anastomosis of ascending aorta to pulmonary artery. Both the Potts and Waterston shunts resulted in excessive pulmonary blood flow, distortion of the pulmonary artery, and problems during subsequent complete ToF repair. Therefore, they are no longer used.
- *Modified BT shunt:* Professor Marc deLeval modified the original BT shunt using an interposition conduit between subclavian artery and pulmonary artery. It is also known as the modified BT shunt, the deLeval shunt, and the GOS (Great Ormond Street) shunt. It is the most commonly used systemic to pulmonary shunt.

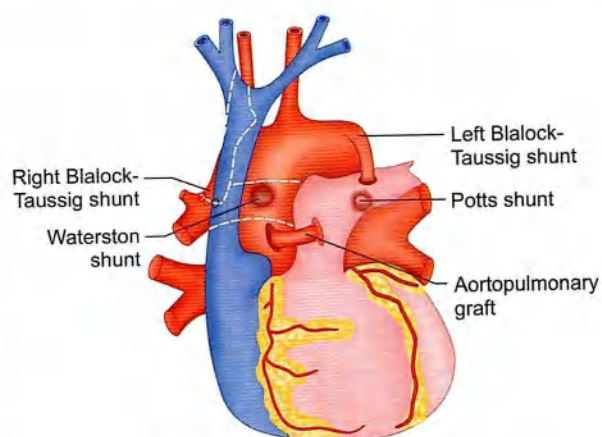


Fig. 2: Systemic to pulmonary shunts.

Definitive Surgery

Earlier only the palliative aortopulmonary shunt operations were done because of high mortality in very young patients. This was later followed by corrective surgery.

However, with the development of deep hypothermic cardiac arrest, low flow hypothermic CPB, and advances in anesthetic management of infants and children and their postoperative care, the morbidity and mortality have decreased dramatically after early one-stage repair. One-stage repair is preferred as it avoids second surgery and eliminates complications of shunt operations. Systemic-to-pulmonary artery shunt is indicated in patients in whom the risk of complete repair is considered greater than the cumulative risk in two-stage repair.

Timing of Surgery

Most centers advocate correction surgery in asymptomatic patients between 4 and 6 months of age, whereas some centers prefer to operate in the early neonatal period and others wait until 1 year of age. Symptomatic patients or those refractory to medical management require urgent surgery irrespective of the age.

Repair at older age is associated with complications of both the palliative and definitive surgery and thus increases long-term morbidity.

Repair

Complete surgical repair involves closure of the VSD and relief of the RVOTO. A median sternotomy approach is used with CPB. There are two surgical approaches:

1. Transventricular approach which involves right ventriculotomy at the infundibulum which allows exposure of the VSD and patch closure
2. Transatrial approach through which the VSD and RVOTO can be approached. Muscle resection is performed to relieve the RVOTO.

Approximately 5% of the patients with ToF have abnormal coronary artery which may modify the surgical approach for repair. The most common is the origin of left anterior descending artery from the right coronary artery.

Q. How will you evaluate these patients preoperatively?

Ans. In addition to the usual pediatric anesthetic evaluation, relevant information should be elicited from the parent as mentioned in the clinical features of ToF.

History

It is important to determine the following:

- Whether the child has normal or impaired functional status?
- Are they gaining weight appropriately? Look for signs of cardiac cachexia.
- Does the child have signs and symptoms of right heart failure (sweating, tachypnea, poor feeding, and recurrent respiratory infections)?
- How is the cyanosis progressing? What is the frequency of cyanotic spells?

- These patients are particularly prone to recurrent respiratory tract infection after palliative shunt surgery secondary to increased pulmonary blood flow and altered lung compliance. Recent upper respiratory tract infection or pneumonia should be ruled out. If a patient suffers from upper respiratory tract infection, surgery may be deferred for a few days till symptoms improve. Failure to do so may result in increased airway reactivity and increase the likelihood of postoperative respiratory complications.
- Other associated congenital anomalies should be identified and relevant knowledge regarding the same and their anesthetic implications should be understood prior to the conduct of anesthesia.
- Previous surgical interventions, if any. Look at the anesthesia records for any problems encountered. If patient has undergone a BT shunt, then arterial line should not be placed on the same side as the information may not be accurate.
- What medications the patient is on at present?
- Lastly, family history of anesthetic difficulties.

Examination

Cyanosis and clubbing may be present in infants older than 6 months. A loud murmur of pulmonary stenosis is audible over the pulmonary area.

Investigations

- *Chest X-ray:* It may reveal a classic boot-shaped heart.
- *Echocardiography and Doppler:* It is a noninvasive tool to assess intracardiac anatomy, blood flow patterns, and estimates of physiologic data.
- *Cardiac catheterization* remains the gold standard for assessing anatomy and physiologic function in CHD. It is indicated before the repair of ToF in patients with previous palliation and when aortopulmonary collaterals, coronary artery, and pulmonary artery branching abnormalities are suspected. As an anesthetist, knowledge of the following catheterization data is important for any case with CHD:
 - Location, size, and direction (left to right or right to left) of intracardiac shunting
 - Pulmonary and systemic arterial pressures
 - Ventricular and atrial pressures with specific attention to left and right ventricular end-diastolic pressure
 - Oxygen saturation data
 - Cardiac chamber size
 - Pulmonary vascular resistance (PVR)
 - Valve anatomy and function
 - Anatomy, location, and function of previously created shunts

- Anatomic distortion of systemic or pulmonary arterial vessels, especially as it relates to previously placed shunts
- Coronary artery anatomy.
- **Full blood count:** Polycythemia and raised white cell count may be suggestive of infection.
- **ECG:** Right axis deviation secondary to RV hypertrophy
- **Arterial blood gas (ABG):** Metabolic acidosis, hypoxemia, and normal CO₂ tension.
- **X-ray chest:** Pulmonary oligemia due to decreased pulmonary arterial flow. Boot shaped heart with an upturned cardiac apex due to right ventricular hypertrophy and concave pulmonary arterial segment may not be seen in many infants.

Q. What are the anesthetic considerations for this child undergoing modified BT shunt surgery?

Ans.

Premedication: The goal of premedication is to achieve adequate sedation maintaining respiratory and hemodynamic stability allowing for easy separation from parents in the young and older children to reduce anxiety.

It can be administered orally or parenterally. Oral premedication is preferred and parenteral route is usually reserved for children who refuse or are not willing to take oral premedication. Midazolam 0.5 mg/kg orally can be given about 20 minutes prior to the induction of anesthesia. Ketamine 5–10 mg/kg orally along with midazolam 0.5–1 mg/kg is sufficient in children who have undergone previous surgical interventions as they get tolerant to midazolam. It can be administered intramuscularly in a dose of 5–8 mg/kg along with glycopyrrolate 10 µg/kg. Midazolam can be administered in a dose of 0.1 mg/kg.

Operating room preparation: Meticulous attention should be paid to make sure that syringes and intravenous tubing is free from air bubbles to prevent paradoxical air embolism. Resuscitative drugs such as calcium gluconate, sodium bicarbonate, atropine, phenylephrine, lignocaine, and epinephrine should be ready for administration. An inotropic infusion, usually adrenaline or noradrenaline, should be ready for administration in high-risk cases. For all pediatric patients, certain anesthetic drugs such as thiopental, ketamine, succinylcholine, and atropine should be kept ready for use on an emergency basis because of the potential for airway reactivity, hypotension, and bradycardia during anesthetic induction.

Monitoring: It should be placed prior to induction of anesthesia, however, in a crying child, these can be established soon after the induction of anesthesia. Monitoring includes seven-lead ECG, noninvasive blood pressure (NIBP), pulse oximetry, end-tidal capnometry,

invasive arterial and central venous pressure lines, temperature, and urine output. Arterial line should not be placed on the side of the shunt surgery. End tidal capnometry is useful in identifying a tet spell intraoperatively. As the pulmonary blood flow decreases so does the end tidal CO₂ and this is followed by arterial desaturation.

Conduct of anesthesia: The anesthetic goal is to maintain the SVR and reduce PVR and prevent hypercyanotic episodes intraoperatively. Hypoxia, hypercapnia, and acidosis (both respiratory and metabolic) increase PVR and should be avoided. Patients should be ventilated in such a way that there is minimal increase in PVR. PVR is lowest at or near the functional residual capacity. Positive-end expiratory pressure (PEEP) may increase the PVR by increasing the alveolar pressure; however, in patients with severe pulmonary stenosis, positive pressure ventilation and PEEP does not cause a clinically significant increase in the PVR. Hypercyanotic “tet” spell if occurs under anesthesia can be treated by fluid resuscitation and increasing the SVR with alpha agonists such as phenylephrine or ephedrine. Also, infundibular spasm can be treated with β-blocker:

- Choice of induction depends on the availability of vascular access. If intravenous induction is chosen, then ketamine is favored as it increases SVR. Inhalation induction is often prolonged due to reduced pulmonary blood flow.
- Nitrous oxide and inhalational agent such as halothane can be used. Halothane is preferred as it relieves infundibular spasm during hypercyanotic spell. Volatile anesthetics decrease the SVR and mean arterial blood pressure in a dose-dependent manner. Caution should be exercised alternatively; total intravenous anesthetic technique can be used to maintain anesthesia.
- Opioid-based anesthesia ensures stable hemodynamics.
- Infective endocarditis (IE) prophylaxis should be given preoperatively.
- Pulse oximeter may not be reliable similarly at lower saturations and the end-tidal carbon dioxide readings underestimate PaCO₂, and this discrepancy worsens with hypoxemia. ABG may be useful.
- Euvolemia should be maintained to prevent dynamic RVOTO.
- As soon as the shunt is open, saturations improve dramatically, but diastolic blood pressure reduces drastically and this can precipitate myocardial infarction in an infant.
- Heparin infusion and aspirin are started to maintain shunt patency.

The shunt flow may be assessed:

- Oxygen saturation around 80% implies that flow is good.

- Oxygen saturation >80% implies that increased pulmonary blood flow and congestion can lead to unilateral pulmonary edema on the side of the shunt.
- Oxygen saturation <80% implies poor pulmonary blood flow.

Q. What are the anesthetic considerations for this child undergoing corrective surgery?

Ans. The anesthesia considerations are similar to that mentioned above; the only difference is that patients posted for corrective surgery require CPB with total body heparinization. Following complete repair with no residual or minimal intrapulmonary shunt, the arterial saturation improves to 100%. Protamine should be administered as patient comes off bypass to reverse the effect of heparin. In patients with RV dysfunction due to RV hypertrophy or following ventriculotomy, surgeon may prefer to leave a residual defect at the atrial level to augment the cardiac output at the cost of arterial desaturation until RV function improves. In such case, the patient may have arterial saturation of around 80%. Inotropes may be required intraoperatively and in the postoperative period. If facilities of micro-TEE (transesophageal echocardiography) is available, it can be used for evaluating, volume status, alterations in inotropic strategy, and completeness of postsurgical assessment.

Q. When should we give IE prophylaxis?

Ans. Infective endocarditis has recently been changed and is recommended in all dental procedures that involve manipulation of gingival tissue or the periapical region of teeth, or perforation of the oral mucosa. It is indicated in patients with:

- Previous history of IE
- Nonrepaired cyanotic CHD, including shunts and conduit
- Complete CHD repair within the previous 6 months
- Repaired CHD with residual defects.

It is not recommended in patients undergoing genitourinary or gastrointestinal tract procedures.

Q. Which conduction abnormalities are observed after corrective surgery for ToF?

- Right bundle branch block (RBBB): 80–90% of cases
- Bifascicular block: 15% of cases
- Premature ventricular contractions (PVC): ~50% of cases
- Atrial arrhythmias: common.

Q. What are the anesthetic considerations in a patient with ToF for noncardiac surgery?

Ans. A child may present either with uncorrected surgery or palliative surgery or corrective surgery. Understanding the pathophysiology of the lesion, its hemodynamic

effects, and pharmacology of anesthetic drugs is essential to tailor the anesthetic for the child. Detailed history and evaluation and investigation as mentioned earlier should be done depending on the type of defect and the nature of noncardiac surgery. Cardiology referral may be needed.

Evaluation of the child's heart and lungs:

- *Cardiac function:* Child's activities—sweats when nursing and slow to finish bottle.
- Rule out congestive cardiac failure: Puffy eyes, enlarged liver (lungs may sound clear), previous hospitalizations for the same, and current treatment, if any.
- *Effect of cyanotic spells:* Passing out (or fainting) indicates very serious R to L shunt.
- Dead space (V_d/V_t) may be very high as the lungs are not well perfused and most of it may be in zone 1. This may be exaggerated by hypovolemia, high pressure during intermittent positive pressure ventilation (IPPV), PEEP, and positioning the child.

Appropriate tailoring of the anesthetic:

- Oral premedication sedation; midazolam with ketamine is a good option
- IE prophylaxis
- Promoting L to R shunt or reducing R to L shunt
- Reducing PVR
- Maintaining SVR and blood pressure
- Maintaining contractility of the heart
- Avoid hypoxia, hypercarbia, acidosis, hyperthermia, pain, and inadequate depth of anesthesia
- Maintain hydration, especially if they are polycythemic
- IV induction in the presence of R to L shunt may be faster, whereas inhalation may be slower secondary to poor pulmonary flow and dilution from the right side
- Avoid air bubbles in syringes and infusions.

If palliative shunt is in place:

- Consider appropriate site for monitoring NIBP and invasive pressure monitoring, if required
- Antibiotic prophylaxis
- Maintain SVR to promote pulmonary blood flow.

Postoperative pain management:

- For corrective or palliative surgery, the child is usually ventilated and so opioids can be used.
- For noncardiac procedures, appropriate analgesia reduces stress on the child and hence reduces cyanotic spells. Parent/nurse-controlled analgesia; regional blocks should be considered for analgesia. Paracetamol and NSAIDs are suitable options.

■ SUGGESTED READING

1. Gabriele B, Maurizio B, Marco B. Does pharmacological therapy still play a role in preventing sudden death in surgically treated tetralogy of Fallot? *Mini Rev Med. Chem.* 2018;18(6):490-4.

2. Gregory A. Gregory Paediatric Anaesthesia, 4th edition. Philadelphia: Churchill Livingstone; 2002.
3. Habib G, Lancellotti P, Antunes MJ, Bongioanni MG, Casalta JP, Del Zotti F, et al. 2015 ESC Guidelines for the management of infective endocarditis: The Task Force for the Management of Infective Endocarditis of the European Society of Cardiology (ESC). Endorsed by: European Association for Cardio-Thoracic Surgery (EACTS), the European Association of Nuclear Medicine (EANM). *Eur Heart J*. 2015;36(44):3075-128.
4. Kaplan JA, Reich DL, Lake CL, Konstadt SN. Kaplan's Cardiac Anesthesia, 5th edition. Philadelphia: Saunders; 2006.
5. Libby P, Bonow RO, Mann DL, Zipes DP. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, 8th edition. Philadelphia: Elsevier Science; 2007.
6. Medscape (2015). Pettersen M, Kulkarni A. Tetralogy of Fallot with pulmonary atresia. [online] Available from: <http://emedicine.medscape.com/article/899368-overview>. [Last accessed on February, 2020].
7. Medscape (2017). Singh VN, Jacobs JP. Tetralogy of Fallot with pulmonary stenosis. [online] Available from: <https://emedicine.medscape.com/article/2063480-overview>. [Last accessed on February, 2020].
8. Medscape (2018). Spektor M, Donson DA. Tetralogy of Fallot (ToF) in adults. [online] Available from: <https://emedicine.medscape.com/article/2035949-overview>. [Last accessed on February, 2020].
9. Motta P, Miller-Hance WC. Transesophageal echocardiography in tetralogy of Fallot. *Semin Cardiothorac Vasc Anesth*. 2012;16(2):70-87.

Anesthetic Considerations in a Patient with Patent Ductus Arteriosus

Reshma Ambulkar, Vandana Agarwal, Malini Joshi

You are asked to evaluate a 22-day-old neonate who is posted for surgery next week. The baby was born preterm at 32 weeks of gestation and she was diagnosed at birth to have a murmur. She is referred to a cardiothoracic center. She weighs 1.2 kg.

Q. Enumerate the common left-to-right shunts.

Ans. The common left-to-right shunts are as follows:

- Atrial septal defect (ASD)
- Ventricular septal defect (VSD)
- Patent ductus arteriosus (PDA).

Q. What are the common complications associated with congenital heart disease?

Ans.

- Infective endocarditis
- Cardiac arrhythmias
- Complete heart block
- Thromboembolism
- Polycythemia.

Q. What is a patent ductus arteriosus?

Ans. Patent ductus arteriosus is a congenital heart disease where there is a persistent vascular structure connecting

the proximal descending aorta to the main pulmonary artery (**Fig. 1**).

Q. Discuss normal fetal circulation.

Ans. Umbilical vein carries oxygenated blood and divides into two branches—(1) portal sinus and (2) ductus venosus. Ductus venosus bypasses the liver and enters the inferior vena cava (IVC), whereas the portal sinus enters the liver. The blood then enters the right atrium, where majority of the blood enters the left atrium through the foramen ovale. From the left atrium, blood passes to the left ventricle and subsequently supplies the brain and the heart. Part of the blood which enters the right atrium mixes with blood from superior vena cava (SVC) and enters the right ventricle. From the right ventricle, blood enters the pulmonary artery, but because of high pulmonary vascular resistance (PVR), blood is shunted to the aorta via the ductus arteriosus (DA). Mixing of saturated and desaturated blood takes place and this supplies the lower body.

Q. What changes occur in fetal circulation at birth?

Ans. When the newborn baby takes the first breath, there is a sharp drop in the intrapleural pressures which may be as low as -25 to -50 mm Hg. This leads to lung expansion and a dramatic drop in the PVR with a substantial increase in the pulmonary blood flow. There is a drop of the right atrial pressure below the left atrial pressure causing the functional closure of foramen ovale. Also, the umbilical cord clamping increases the systemic vascular resistance (SVR) which maintains the left atrial pressure. With the rise in PaO_2 from the low fetal levels, the flow through the DA decreases and eventually leads to closure (**Flowchart 1**).

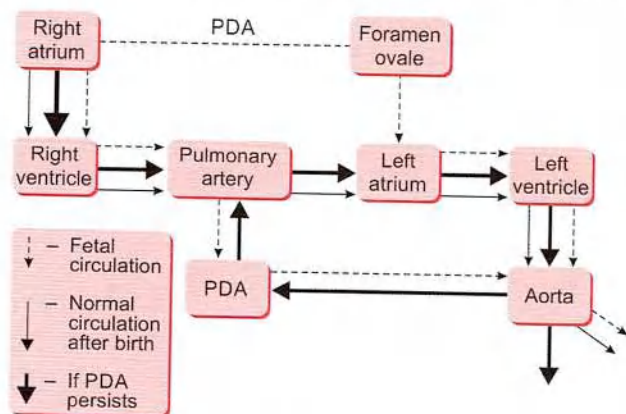
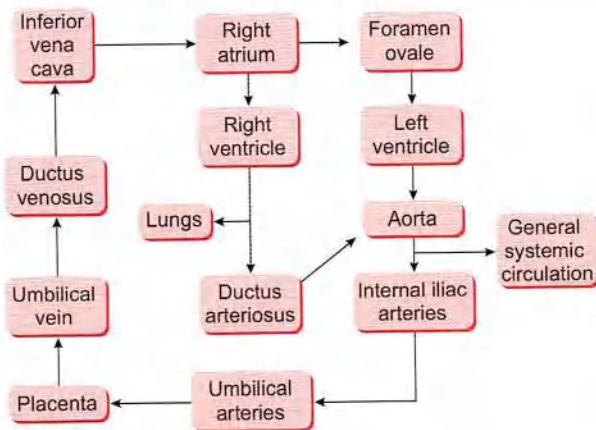


Fig. 1: Patent ductus arteriosus (PDA).

Flowchart 1: Fetal circulation.

**Q. Why is the size of PDA important?**

Ans. The clinical significance of the PDA and the physiological impact caused by it depend largely on its size as it decides the magnitude of the left-to-right shunt. The other factors which determine the flow through the ductus are the pressure gradient from aorta to pulmonary artery, PVR, and SVR.

Q. Discuss the embryological development of the heart and the DA.

Ans. Embryology of heart: Cardiac formation is almost complete by 55–60 days postconception but by the age of 2–3 years, cardiovascular system essentially is that of a fit adult.

- Cardiovascular system starts to develop at 3 weeks of gestation
- Beating of the heart starts soon after 3 weeks of gestation
- Chamber formation is as early as 5 weeks
- Valve formation is delayed till about 7 weeks.

Embryology of DA: The proximal portions of the sixth pair of embryonic aortic arches form the proximal branch of pulmonary arteries, and the distal portion of the left sixth arch forms the DA, connecting the left pulmonary artery with the left dorsal aorta.

Q. What are the causes and risk factors associated with PDA?

Ans. Prematurity: The increased incidence of PDA is due to developmental immaturity in premature babies.

Genetic factors: Genetic mechanism responsible for PDA is autosomal recessive inheritance with incomplete penetrance. PDA is associated with several genetic syndromes, including trisomy 21, single-gene mutations (Carpenter's syndrome), and X-linked mutations (incontinentia pigmenti).

Rubella infection: There is a good association between rubella infection during the first trimester of pregnancy and increased incidence of PDA.

Teratogens: Drugs such as amphetamine and phenytoin when consumed by mother are associated with increased incidence of PDA.

Poorly controlled diabetes in mother also predisposes to birth of babies with PDA.

Q. How would you define prematurity?

Ans. When the gestational age is <37 weeks, the baby is termed as premature.

- *Moderately premature:* 31–36 weeks of gestation
- *Severely premature:* <31 weeks of gestation
- Prematurity is common and occurs in 5–10% of live births.

Significance: Due to immature development of organs, there is a higher incidence of mortality and morbidity in these babies and is associated with the following complications:

- Respiratory failure
- Recurrent pneumonia
- Apnea spells
- Respiratory distress syndrome
- Hyaline membrane disease
- Bronchopulmonary dysplasia
- PDA
- Malnutrition
- Necrotizing enterocolitis
- Poor gastrointestinal (GI) motility and function
- Anemia
- Growth failure
- Metabolic disturbances
- More prone to infection
- Kernicterus
- More prone to hypothermia
- Fractures
- Failure to thrive
- Social—child neglect
- Hypoxic ischemic encephalopathy
- Retinopathy.

Q. What causes the closure of DA? When does it occur?

Ans. The patency of the DA during fetal life is due to low fetal oxygen tension and cyclooxygenase-mediated products of arachidonic acid metabolism, primarily prostaglandin (PGE₂), and prostacyclin (PGI₂). High levels of PGE₂ and PGI₂ are due to the production by placenta and decreased metabolism by the fetal lungs. PGE₂ and PGI₂ interact with ductal prostanoid receptors resulting in the vasodilatation of DA. After birth, the abrupt increase in oxygen tension inhibits ductal smooth muscle potassium channels resulting in calcium influx and ductal constriction. PGE₂ and PGI₂ levels also fall because of increased metabolism by the lungs and cessation of production by the placenta. The medial smooth muscle fibers in the ductus contract resulting in lumen obliteration of the DA. Functional closure of the

ductus occurs within 24–48 hours of birth and within 2–3 weeks causing fibrosis leading to a permanent closure.

Q. Discuss the pathophysiologic consequences of left-to-right shunting.

Ans. The magnitude of shunting depends on the resistance to flow through the ductus. The elasticity of the ductus as well as the length and diameter would decide the resistance to the flow. The other important factor is the pressure gradient between the two vessels—(1) the aorta and (2) the pulmonary artery. The left-to-right shunting of blood through the ductus results in pulmonary overcirculation and left heart volume overload. The increased pulmonary circulation results in decreased lung compliance and increased work of breathing. In older patients, pulmonary edema may result. The volume overload of the left heart results in an increase in end-diastolic pressures of both the atria and the ventricles. The left ventricle initially compensates by increase in stroke volume but left ventricle hypertrophies overtime. There is also an accompanying increase in the heart rate due to increased levels of catecholamines which is a part of neuroendocrine adaptation. All the above factors may result in subendocardial ischemia owing to a mismatch in oxygen demand and supply.

Q. What is Eisenmenger's syndrome?

Ans. The pulmonary vasculature undergoes changes with time owing to high volume as well as pressure. There is a progressive increase in PVR due to hypertrophy and fibrosis in the vascular system resulting in luminal obliteration in pulmonary arterioles and capillaries. When the PVR exceeds SVR, shunting of blood reverses and changes from left to right to right to left. This is called Eisenmengerization and the syndrome is called Eisenmenger's syndrome.

Q. What is the clinical presentation of a patient with PDA?

Ans. The child presents with the following and rarely the child may be asymptomatic:

- Poor growth
- Breathlessness
- Easy fatigability
- Tachycardia
- Frequent lung infections.

The physical examination reveals:

- Prominent or bounding peripheral pulse.
- Systolic hypertension and low diastolic pressure due to increased left ventricular stroke volume and flow from aorta to pulmonary circulation.
- The first heart sound (S_1) is normal. The second heart sound (S_2) often is masked by the murmur.
- Continuous machinery murmur is best heard along the upper left sternal border, radiating down the left side of the sternum and into the back. A thrill may be present.

- *Apical flow rumble:* The pulmonary blood flow exceeds the systemic blood flow causing a left-sided volume overload which leads to increased blood flow from the left atrium to left ventricle across the mitral valve causing a flow rumble.
- *Cyanosis:* Babies with Eisenmenger's syndrome present not only with cyanosis but differential cyanosis, which is cyanosis and clubbing of the toes but not the fingers because the right-to-left ductal shunting is distal to the subclavian arteries.

Q. How would you investigate such a child?

Ans. Chest radiography shows:

- Cardiomegaly with left atrial and ventricular enlargement is present.
- With marked pulmonary overcirculation, signs of pulmonary edema are seen.
- In elderly individuals, calcification of PDA may be seen.
- Rarely, it may be normal.

Electrocardiogram:

- Sinus tachycardia or atrial fibrillation
- Small PDA—the ECG (electrocardiogram) findings may be normal
- Larger PDA—left ventricular hypertrophy
- In the neonate, especially the premature neonate with a large PDA, T-wave inversion, and ST segment depression may be present suggesting ischemia or a supply-demand mismatch.

Doppler echocardiography:

- It helps to confirm the diagnosis.
- It is useful in describing the PDA as silent, small, moderate, or large.
- Presence of reversal of forward flow in the descending aorta during diastole, left atrial or ventricular dilation, due to overcirculation of the pulmonary vascular bed and a chronic hyperdynamic state, and left-to-right flow across the intra-atrial septum are indicative of a large shunt.
- The ratio of the left ventricular output to SVC flow is directly proportional to the ductus flow and, when ≥ 4 , may indicate hemodynamic significance.
- A left atrial to aortic root (LA/Ao) ratio is considered abnormal if >1.5 . For comparison, the ductus is considered widely patent when the LA/Ao ratio >1.6 , and closed when the ratio is 1.17. If a cutoff of 1.4 is used, sensitivity of the LA/Ao ratio drops from 88% to between 50 and 79%.
- *Color Doppler:* It is a very sensitive modality used in detecting the existence of a PDA and the amount of shunting.

M-mode echocardiography:

- It is used to measure the cardiac chamber sizes and quantify left ventricular systolic function.

- **Small PDA:** Chamber sizes are usually normal, although mild left atrial and/or left ventricular enlargement may be present.
- **Moderate or large PDA,** and the left atrium and the left ventricle are enlarged.

Biomarkers

The N (amino)-terminal prohormone of B-type natriuretic peptide (NT-proBNP) and mature B-type natriuretic peptide (BNP) may be useful in detecting a hemodynamically significant PDA. BNP is secreted and released by the ventricular myocardium when under stress from either increased volume or pressure. NT-proBNP, BNP, and cardiac troponin rise with increase in echocardiographic markers of PDA, and decrease following the closure of the ductus. They are likely to be of most benefit when the diagnosis of PDA is confirmed with echocardiography, but serial echocardiography to determine hemodynamic significance is unavailable, or to shorten a course of pharmacologic treatment for a ductus.

Biosensors

Bioengineering and analytical techniques have also been developed to monitor or detect PDA in preterm infants. Various approaches include interpretation of pulse oximetry information (perfusion index and plethysmography), interpretation of transthoracic electrical signals (impedance, cardiometry, velocimetry, and bioelectrical), regional oxygenation (near-infrared spectroscopy), alterations in skin microcirculation (side stream dark-field imaging and reflectance spectrophotometry), and resonance Raman spectroscopy.

Cardiac Catheterization

- Therapeutic catheterization is the treatment of choice for PDA. It helps in complete diagnostic evaluation of hemodynamics of the patient as it is important to assess the PVR and the amount of shunting before transcatheter closure.
- It also helps in assessing the response of vasodilating agents such as oxygen, nifedipine, prostacyclin, sildenafil, and nitric oxide in patients with raised PVR.

Q. Enumerate the complications associated with PDA.

Ans.

- **Congestive heart failure:** In moderate-to-large PDA, congestive heart failure due to pulmonary overcirculation and left heart volume overload can occur.
- **Atrial flutter or fibrillation:** Heart failure is frequently associated with atrial flutter or fibrillation in adults.
- **Hypertensive pulmonary vascular disease:** Secondary to long-standing pressure and volume overload in the pulmonary circulation.

- **Endarteritis:** Vegetations usually occur in the pulmonary artery end of the ductus and embolic events are usually of the lung rather than the systemic circulation.
- **Aneurysm of DA:** Ductal aneurysm most commonly presents in infancy but has also been reported in adults and may develop after infective endarteritis, surgical closure, or transcatheter coil occlusion.
- **Rare complications:** Dissection and/or spontaneous rupture of a dilated pulmonary artery in PDA associated with pulmonary hypertension.

Q. What are the treatment options for PDA?

Ans. Medical management consists of attempts to close the PDA and treatment of complications.

- **Indomethacin:** Intravenous indomethacin is effective in closing a PDA if it is administered in the first 10–14 days of life. Indomethacin is a potent inhibitor of prostaglandin-forming cyclooxygenase, hence there is a decrease in the synthesis of prostaglandins which leads in ductal closure:
 - **Dose:** 0.1–0.2 mg/kg, three doses every 12 hourly
 - **Adverse effects:** Decreased mesenteric, renal, and cerebral blood flow
- **Ibuprofen:**
 - **Dose:** IV 20 mg/kg followed by 10 mg/kg 24 hours apart for total three doses
 - **PO:** 10 mg/kg followed by 5 mg/kg 24 hours apart for total three doses
- **Acetaminophen:** It reduces prostaglandin synthesis by a different mode of action than most nonsteroidal anti-inflammatory drugs (NSAIDs).
 - **Dose:** IV or PO—7.5, 10, or 15 mg/kg per dose every 6–8 hours for 3–7 days
- **Cautious fluid restriction**
- **Antiarrhythmia drugs:** Treatment of atrial fibrillation or flutter
- **Vasodilators:** Patients with PDA and pulmonary vascular disease who are considered unacceptable candidates for definitive closure are managed with pulmonary vasodilating agents such as PGI₂, calcium channel blockers, endothelin antagonists, and phosphodiesterase type V inhibitors.
- **Other drugs:** Diuretics and digoxin (avoid in small preterms).

Surgical Management Options

Transcatheter closure: Advance a catheter or delivery sheath across the DA from either the pulmonary artery or the aorta and position a closure device in the ductus to occlude it.

Success rate: Complete closure rates >90–95%.

Complications of this procedure are:

- Bleeding at the catheterization site
- Rupture of blood vessels

- Tachyarrhythmias
- Bradyarrhythmias
- Vascular occlusion
- Inappropriate deployment of the device
- Migration of the device
- Incomplete closure of the ductus.

Surgical closure: Surgical ligation or division of the PDA remains the treatment of choice for the very large PDA or hemodynamically significant PDA that results in cardiac dysfunction, renal failure, or respiratory failure. When the PDA does not permit ligation due to inadequate length, patch closure on cardiopulmonary bypass is necessary.

Success rate: Complete closure rates between 94 and 100%. Complications of this procedure are:

- Bleeding
- Injury to the recurrent laryngeal nerve
- Disruption of the thoracic duct with resultant chylothorax
- Injury to the vagus nerve
- Ligation of the left pulmonary artery
- Ligation of the descending aorta or other arterial structures within the chest
- Pneumothorax
- Infection
- **Mortality:** 0–2%.

Q. Discuss the anesthetic considerations in a patient of PDA.

Ans. Today cardiac surgery is a proven and effective treatment for children with congenital heart defects as the survival of these patients are unequivocally influenced by surgical therapy. If the shunt is nonrestrictive and does not impede blood flowing freely in each direction, then the main determinant of blood flow is the resistance of the pulmonary and systemic vascular beds. Left-to-right shunts result in increased pulmonary blood flow and hence three pathophysiologic problems:

1. Volume overload of the pulmonary circulation.
2. Increased cardiac work of the left ventricle which is required to increase the stroke volume and the heart rate to ensure adequate systemic perfusion.
3. Excessive pulmonary blood flow resulting in progressive elevation in PVR.

There are concerns about the increased workload on immature heart of the baby because of the following reasons:

- Relatively noncompliant
- Relatively restricted ability to change stroke volume
- Plateau of pressure–volume curve reached early
- Cardiac output more rate-dependent.

Persistence of large left-to-right shunts may outstrip the capacity of the left heart to maintain adequate systemic

perfusion and result in congestive heart failure. The chronic effects of congenital heart disease are a consequence of the imposed hemodynamic stress of the defect and sequelae after interventional cardiac surgery which alters normal growth and development of the cardiovascular system as well as other organ systems throughout life. Multidisciplinary team-oriented approach involving cardiac surgeons, cardiologists, anesthesiologists, intensivists, and nurses working as a team in preparing the patient and the family for surgery and postoperative recovery is ideal and beneficial. Anesthetic management should be based on sound physiology principles.

History: Detailed history regarding birth history and maternal drug history:

- Cyanosis
- Exercise tolerance
- Feeding history
- Medications
- Previous cardiac/other surgery.

Proper management of the physiological abnormalities is more important than the choice of specific anesthetic and pharmacological approaches.

Premedication: The goal of premedication is to achieve adequate sedation in a nontraumatic fashion and maintain respiratory and hemodynamic stability. In a premature neonate, sedation is not required.

Monitoring

- **Preinduction:**
 - Precordial stethoscope
 - Pulse oximetry
 - ECG (II +/–V5)
 - Noninvasive blood pressure
- **Preincision:**
 - Invasive blood pressure
 - Hourly urine output measurement
 - Esophageal stethoscope
 - Temperature monitoring
 - Central venous pressure measurement may or may not be used
- **Other monitors may be used:**
 - Doppler transducer
 - EtCO₂
 - Arterial blood gas (ABG)
 - Inspired gas monitoring.

Appropriate antibiotics should be given as prophylaxis against bacterial endocarditis.

Anesthesia induction: Preoxygenate with 100% O₂. Analgesia with fentanyl: 1–3 µg/kg bolus. Inhalational induction with sevoflurane which decreases SVR and improves systemic

blood flow by decreasing the magnitude of left-to-right shunt. Intubate with the appropriate-sized endotracheal tube (ETT). Ventilation: Positive pressure ventilation is well tolerated in these patients.

Maintenance of anesthesia: Lower doses of fentanyl (1 µg/kg). Caution about high-dose opioids as they are not well tolerated in neonates and cause hypotension. Muscle relaxation can be with either vecuronium or pancuronium. Maintenance of anesthesia with air—O₂ mixture and volatile anesthetics.

Reversal: Postoperative ventilation may be needed due to the prematurity of the baby.

Other concerns:

- Blood loss, if control of PDA is lost during ligation
- Hypoglycemia is a frequent concern in neonates during the perioperative period
- Hypothermia as preterm infants have impaired thermal regulatory ability
- *Systemic hypertension in the postoperative period:* Ligation of PDA is associated with hypertension, which

can be managed with infusion of vasodilating drugs such as sodium nitroprusside.

In summary, anesthetic goals are:

- To decrease the shunt flow
- To maintain cardiovascular and respiratory stability
- To provide adequate tissue perfusion and oxygenation.

Q. In what scenarios would you like to keep DA open and how will you do it?

Ans. In transposition of the great vessels (the pulmonary artery and the aorta), a DA may need to remain open as it is the only way that oxygenated blood can mix with deoxygenated blood. Prostaglandins are used to keep the PDA open.

■ SUGGESTED READING

1. Allman KG, Wilson IH. Oxford Handbook of Anaesthesia, 4th edition. Oxford, United Kingdom: Oxford Press; 2016.
2. Miller R, Eriksson L, Wiener-Kronish J, Cohen N, Young W. Miller's Anesthesia, 8th edition. Philadelphia: Saunders Elsevier; 2015.
3. Yao FSF. Yao and Artusio's Anesthesiology, 8th edition. Philadelphia: Wolters Kluwer; 2016.

Permanent Pacemaker

Sumitra Ganesh Bakshi, Vijaya Prakash Patil, Sheila Nainan Myatra

A 65-year-old patient with a pacemaker comes for total knee replacement. The patient gives a history of hospitalization for myocardial infarction 4 years back during which a pacemaker was inserted. The patient is asymptomatic now, however, his daily activities are restricted in view of his arthritis. Describe the perioperative management of this patient.

Q. What are the indications for a permanent pacemaker?

Ans. Artificial pacemaker is indicated for treatment of symptomatic bradycardia of any origin. Indications of permanent pacemaker implantation are as follows:

Acquired atrioventricular (AV) block:

- **Third-degree AV block:**
 - Bradycardia with symptoms
 - Postcardiac surgery AV block not expected to resolve
 - Neuromuscular disease with AV block
 - Escape rhythm <40 bpm or asystole >3 seconds.
- **Second-degree AV block:** Permanent or intermittent symptomatic bradycardia.
- **After myocardial infarction:**
 - Persistent second-degree or third-degree block
 - Infranodal AV block with left bundle branch block (LBBB)
 - Symptomatic second- or third-degree block.
- **Bifascicular or trifascicular block:**
 - Intermittent complete heart block with symptoms
 - Type II second-degree AV block
 - Alternating bundle branch block.
- **Sinus node dysfunction:**
 - Sinus node dysfunction with symptoms as a result of long-term drug therapy
 - Symptomatic chronotropic incompetence.
- **Hypertensive carotid sinus and neurocardiac syndromes:**
 - Recurrent syncope associated with carotid sinus stimulation
 - Asystole of >3 seconds duration in absence of any medication

• Prevention and termination of arrhythmias by pacing:

- Symptomatic recurrent supraventricular tachycardia (SVT) that is reproducibly terminated by pacing when catheter ablation and/or drugs fail to control the arrhythmia or produce intolerable side effects
- Sustained pause-dependent ventricular tachycardia (VT), with or without QT prolongation.

A small minority of patients with bifascicular block may develop transient or established complete heart block. The risk of progression from bifascicular block to complete heart block in asymptomatic patients is very small. Insertion of temporary transvenous pacemaker before anesthesia is not necessary; an external pacemaker can be made available.

In summary: Indications of permanent pacemaker includes:

- Symptomatic bradycardia—escape rhythm <40 bpm or asystole >3 seconds
- **Acquired AV block:**
 - Postmyocardial infarction/surgery not expected to resolve
 - Symptomatic (syncope)
- Sinus node dysfunction/hypertensive carotid sinus and neurocardiac syndromes with symptoms.

Q. What are the various types of pacemakers?

Ans. Pacemakers can be classified as per the method of insertion, location of leads, etc:

- Transvenous
- Transcutaneous
- Epicardial
- Transesophageal

The original nomenclature by Intersociety Commission for Heart Disease (ICHD) resources involved a three-letter code. This code has been extended to five letters. **Table 1** gives these generic codes for pacemaker.

TABLE 1: Generic codes of pacemaker.

I. Chamber paced	II. Chamber sensing	III. Response	IV. Programmability	V. Tachycardia
O—none	O—none	O—none	O—none	O—none
A—atrium	A—atrium	I— inhibited	C—com- municating	P—pacing
V— ventricle	V— ventricle	T— triggered	P—simple program- mable	S—shock
D—dual (A + V)	D—dual (A + V)	D—dual (I + T)	M—mul- tiple program- mable	D—dual (P + S)
			R—rate modulation	

Modern-day pacemakers are programmable into three following modes:

1. Asynchronous pacing or fixed rate (AOO, VOO, and DOO) paces at preset rate that is independent of the inherent heart rate. The most dangerous complication is ventricular fibrillation due to R on T phenomenon.
2. Single-chamber demand pacing (AAI and VVI) paces at a preset rate when spontaneous heart rate falls below the preset rate. The firing of the pacemaker is inhibited by electrical activity in the chamber sensed.
3. Dual-chamber AV sequential pacing requires two pacemaker leads, one in the atrium and one in the ventricle. The atrium is stimulated to contract first, and after an adjustable PR interval, the ventricle is stimulated to contract. Dual-chamber pacing is indicated when ventricular pacing alone cannot maintain adequate cardiac output and atrial pacing alone cannot be done as

in third-degree AV block. Major advantage is the ability to increase the cardiac output and reduce the incidence of atrial fibrillation.

Q. Explain some common terminologies used with a pacemaker.

Ans. The common terminologies used with a pacemaker are as follows:

- **Pulse generator:** It includes the energy source (battery) and electric circuits for pacing and sensory function. Mercury-zinc batteries that were used in the early days had a short useful life (2–3 years). Currently, lithium iodide batteries are being used which have longer life (5–10 years) and high energy density.
- **Leads:** Insulated wires connecting pulse generator and electrodes.
- **Electrode:** An exposed metal end of the lead in contact with the endocardium or epicardium.
- **Unipolar pacing:** There is one electrode, the cathode (negative pole) or active lead (**Fig. 1**). Current flows from the cathode, stimulates the heart, and returns to the anode (positive pole) on the casing of pulse generator via the myocardium and adjacent tissue to complete the circuit. A unipolar pacemaker is more likely to pick up extracardiac signals and myopotentials. Pacemaker spikes are large in this pacing (**Fig. 2**).
- **Bipolar leads:** Two separate electrodes, anode (positive pole) and cathode (negative pole), are located close to each other within the chamber that is being paced (**Fig. 1**). As the electrodes are very close, the circuit is small and the possibility of extraneous noise disturbance is less and the signals are sharp. Pacemaker spikes are very small in this type of pacing (**Fig. 3**).

Endocardial pacing: It is also called as transvenous pacing which implies that the leads/electrodes system has been passed through a vein to the right atrium or the right ventricle. It can be unipolar or bipolar.

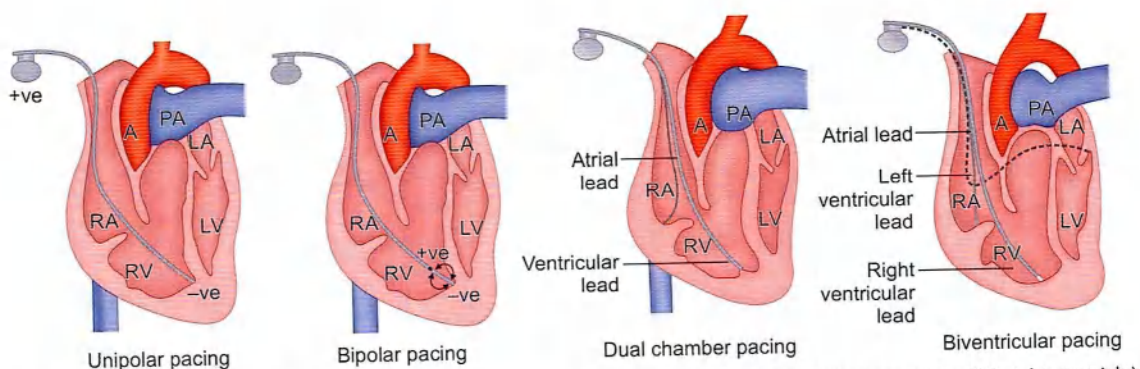


Fig. 1: Different types of pacemakers. (LA: left atrium; LV: left ventricle; PA: posteroanterior; RA: right atrium; RV: right ventricle).

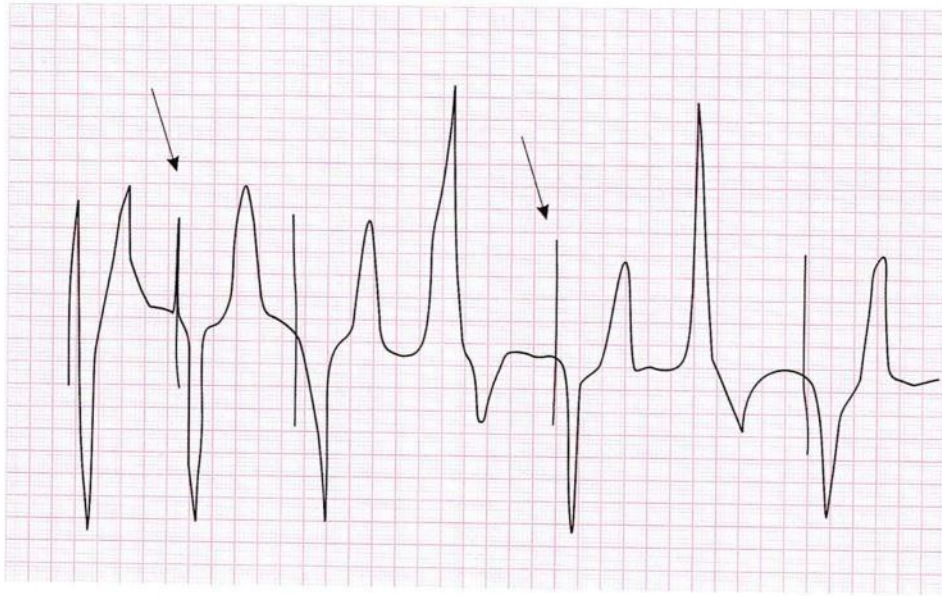


Fig. 2: Unipolar pacing.

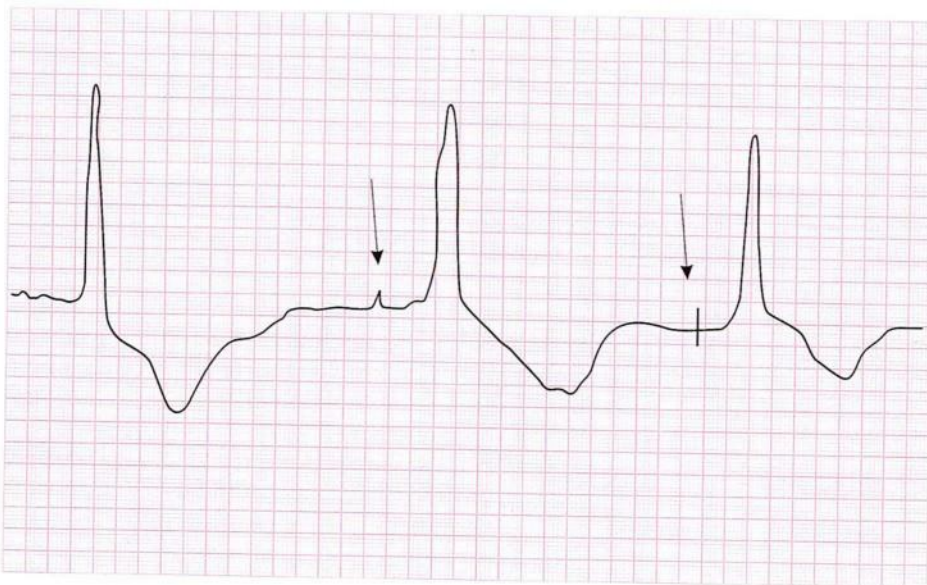


Fig. 3: Bipolar pacing.

Epicardial pacing: This type of pacing is accomplished by inserting the electrode through the epicardium into the myocardium and is generally done following cardiac surgery. This can also be unipolar or bipolar.

Pacing threshold: This is the minimum amount of energy required to consistently cause depolarization and contraction of the heart.

Sensitivity: The minimal voltage level of the patient's intrinsic R wave or P wave that must be exceeded for the pacemaker to sense that R wave or a P wave and to activate

the sensing circuit of the pulse generator, resulting in inhibition or triggering of the pacing circuit.

Hysteresis: It is particularly useful in patients with sick sinus syndrome. This feature allows a longer escape interval after a sensed event, giving the heart a greater opportunity to beat on its own. Here, pacemaker is programmed to upper and lower rate and a programmable lower hysteresis rate.

Runaway pacemaker: It is pacemaker dysfunction characterized by fast and erratic spikes. This occurs with generator dysfunction due to battery failure or damage

produced by the leakage of the tissue fluids into the pulse generator.

Q. What do we understand by programmable pacemaker?

Ans. Our physiological pacemaker, SA node, responds to body's changing demands by increasing or decreasing the heart rate. Patients on modes such as DDD, VVI, and AAI cannot increase or decrease the heart rate according to the metabolic demands. To overcome this problem, recent-generation pacemakers provide flexibility to adapt the device to patient's changing metabolic needs, and are capable of detecting body movements (vibrations), central venous oxygen saturation, respiratory rate, and depth.

Q. What is a biventricular pacemaker? What is cardiac resynchronization therapy (CRT)?

Ans. Cardiac resynchronization therapy devices have been shown to reduce mortality and improve quality of life in patients with heart failure symptoms. Electrical depolarization is normally initiated throughout both ventricles by the His-Purkinje system. In patients with systolic dysfunction with conduction disturbances as manifested by prolonged QRS complex, conduction of the wave of depolarization in the left ventricle is markedly altered. As a result, left ventricular contraction becomes dyssynchronous (septum and lateral wall contracting asynchronously), with resultant decrease in stroke volume, increased wall stress, and delayed relaxation. A biventricular pacemaker, also known as CRT, is a type of pacemaker that paces both the septal and lateral walls of the left ventricle (see Fig. 1). By pacing both sides of the left ventricle, the pacemaker can resynchronize left ventricle to contract in synchrony. This has been shown to be useful in heart failure. CRT devices have at least two leads, one in the right ventricle to stimulate the septum, and another inserted through the coronary sinus to pace the lateral wall of the left ventricle. For patients in normal sinus rhythm, there is also a lead in the right atrium to facilitate synchrony with the atrial contraction. Thus, timing between the atrial and ventricular contractions, as well as between the septal and lateral walls of the left ventricle can be adjusted to achieve optimal cardiac function. Patients with dilated cardiomyopathy, with left ventricular ejection fraction (LVEF) ≤ 0.35 , a QRS complex ≥ 120 ms, sinus rhythm, and are New York Heart Association (NYHA) functional class III or IV, despite maximal medical therapy for heart failure, are candidates for CRT.

Q. What is pacemaker syndrome?

Ans. Pacemaker syndrome, also known as AV dyssynchrony syndrome, represents the clinical consequences of AV dyssynchrony or suboptimal AV synchrony, regardless of the pacing mode. This leads to a variety of clinical signs

including hypotension, syncope, vertigo, lightheadedness, fatigue, and exercise intolerance. Symptoms may vary from mild to severe, and onset may be acute to chronic. The main mechanism behind these symptoms is loss of atrial kick with resultant drop in cardiac output.

Q. How would you evaluate this patient preoperatively?

Ans. Preoperative evaluation includes:

- *Detailed evaluation of the underlying cardiovascular disease* responsible for the insertion of pacemaker
- *Other associated medical problems:* Since a substantial number of these patients suffer from coronary artery disease (50%), hypertension (20%), and diabetes (10%), one should know:
 - Severity of the cardiac disease
 - Current functional status
 - Medications the patient is on
 - The initial indication for the pacemaker
 - Preimplantation symptoms such as lightheadedness, dizziness, or fainting. If these symptoms occur even after the pacemaker insertion, cardiology consultation is needed
 - Bruits and signs of congestive cardiac failure (CCF).

Evaluation of Cardiac Status

Dobutamine stress test: Most commonly performed stress test uses exercise on treadmill and looking for ECG (electrocardiogram) changes when the heart rate is achieved to 80% maximum for the age. In patients with pacemaker without rate modulation, HR is fixed and thus cannot be increased by exercise (chronotropic incompetence). Despite the presence of chronotropic incompetence among pacemaker-dependent patients, there is preservation of both a positive inotropic response to dobutamine with increased myocardial contractility and changes in left ventricular loading conditions leading to increased myocardial oxygen demand. Thus, the test can help in detecting ischemia on the basis of factors independent of the heart rate.

Evaluation of the Pacemaker (Practice Advisory for the Perioperative Management of Patients with Cardiac Rhythm Management Devices)

- Establish whether a patient has a cardiac rhythm management device (CRMD)
- *Focused history:* Details of manufacturer and identification card
- Physical examination for scars and palpating the device (generally, a generator for the epicardial electrodes is kept in the abdomen and over one of the pectoris muscles for the endocardial electrodes).

Investigations

- Routine biochemical and hematological investigations should be performed as indicated on an individual basis.

- Measurement of serum electrolytes (especially K^+) should be performed.
- *A 12-lead ECG:* In atrial pacing, an electrical spike appears before the P wave and the QRS complex is usually normal. In ventricular pacing, there are two spikes, one before the P wave and another preceding the QRS complex.
- X-ray of chest (for visualization of continuity of leads).

Q. How will you evaluate the function of the pacemaker in the preoperative period?

Ans. Assistance from the cardiologist and the manufacturer's representative should be obtained for the purpose.

Most of the information about the pacemaker, such as type of pacemaker (fixed rate or demand rate), time since implanted, pacemaker rate at the time of implantation, and half-life of the pacemaker battery, can be taken from the manufacturer's book kept with the patient. Details of pacemaker function can be elicited from the patient regarding last battery and pulse generator check. 10% decrease in the rate from the time of implantation indicates power source depletion. In patients with VVI generator, if intrinsic heart rate is greater than pacemaker set rate, evaluation of pacemaker function can be done by slowing down the heart rate by carotid sinus massage, while the patient's ECG is continuously monitored. Carotid sinus massage should be avoided in patients with a history suggestive of cerebrovascular disease or carotid artery disease, because the atherosclerotic plaque may embolize to the cerebral circulation. Other methods to slow the heart rate are Valsalva maneuver and the use of edrophonium. Reprogramming the pacemaker is generally indicated to disable rate responsiveness. Patients with implanted cardioverter defibrillator (ICD) should have the tachycardia therapy (antitachycardia pacing and shocks) deactivated before surgery that may require cautery. If the risk of electromagnetic interference (EMI) is high, such as, when the electricity is in close proximity to the generator, alternative temporary cardiac pacing device should be kept ready.

Q. Describe the effect of magnet application on pacemaker function.

Ans. During surgery, EMI from electrocautery and similar devices can seriously interfere with the function of pacemaker working in a demand mode. This can be avoided by converting the pacemaker to a fixed rate asynchronous mode by application of a magnet. Magnet application is an extremely important function. Most pacemakers and ICDs have built-in magnetic reed switches that are designed to switch "ON" or "OFF" circuitry in response to magnets. The magnet is placed over the pulse generator to trigger the reed switch present in the pulse generator resulting

in a nonsensing asynchronous mode with a fixed pacing rate (magnet rate). Magnet-operated reed switches were originally incorporated to produce pacemaker behavior that would demonstrate remaining battery life and sometimes pacing thresholds. Activation of the reed switch shuts down the demand function so that the pacemaker stimulates asynchronous pacing. Thus, magnets can be used to protect the pacemaker-dependent patient during EMI, such as diathermy or electrocautery. Also, if no information is available from the patient about the pacemaker, magnet may identify the particular model with the help of magnet rate, which varies among different manufacturers and thus provide clue for its identification. However, not all pacemaker's switch to asynchronous mode on the application of magnet. The response varies with the model and the manufacturer, and may be in the form of no apparent change in rate or rhythm, brief asynchronous pacing, continuous or transient loss of pacing, or asynchronous pacing without rate response. In an ICD system, magnet cannot convert the pacemaker to asynchronous mode, it only disables tachycardia detection and therapy of the ICD. Clinical magnets, made of ferrous alloy, come in various shapes (ring or doughnut, horseshoe, and rectangle or bar). The site of magnet placement is important since a poorly positioned magnet may not produce the desired effect. Magnets are best placed directly on top of the device. It is advisable to consult the manufacturer to know the magnet response before use. The patient must be connected to an electrocardiograph recorder before the magnet is applied and, remain connected, until after the magnet is removed. The first few paced complexes after magnet application provide information regarding the integrity of the pulse generator and its lead system. A 10% decrease in the magnet rate from the time of implantation indicates power source depletion and is an indicator of end-of-life requiring elective replacement of battery. If magnet application on a pacemaker site does not produce any response on the surface ECG pacing rate or mode, the magnet may be repositioned. If no change is still observed, reasons may be: (1) A depleted pacemaker battery, (2) The pacemaker is programmed to ignore the magnet (St. Jude, Boston Scientific, and Biotronik synchronous mode), and (3) The magnetic field does not reach the device, as in the case of those with deeper (abdominal or submuscular) implants or in very obese patients.

Q. How do you test for the chronotropic response of the paced heart?

Ans. In patients with bundle branch block (except for cases with complete heart block) and endogenous activity, a small dose of adrenaline is given intravenously; and the pacemaker demand mode is reduced—the chronotropic

response of SA node is noted. If the response is adequate, then it can be concluded that the patient is not entirely pacemaker dependent.

Q. How would you reprogram the ICD preoperatively?

Ans. Implanted cardioverter defibrillator can be reprogrammed to disable the antitachycardia function before surgery in operating room where a defibrillator is readily available. A magnet can be placed over the ICD to disable the antitachycardia function in the operating room. The advantage of placing a magnet on an ICD is that the magnet can be easily removed and antitachycardia function is quickly enabled in case of VT/VF (ventricular fibrillation) during surgery.

Q. How do you preoperatively prepare the patient for surgery and anesthesia?

Ans. Preoperative preparation includes the following:

- Determine whether EMI is likely to occur during the planned procedure.
- Determine whether reprogramming the CRMD, i.e., changing to an asynchronous pacing mode or disabling any special algorithms, including rate-adaptive functions, is needed.
- Suspend antitachyarrhythmia function, if present.
- Advise the surgeon performing the procedure to consider use of a bipolar electrocautery system or ultrasonic scalpel to minimize potential adverse effects of EMI on the pulse generator or leads.
- Assure the availability of temporary pacing and defibrillation equipment and drugs for resuscitation.
- Evaluate the possible effects of anesthetic technique on CRMD function and patient CRMD interactions.

Q. Intraoperatively, you can see the pacing spikes but they are not followed by QRS complex or ventricular activity. What has gone wrong? What is pacing threshold and what are the factors affecting it?

Ans. Pacing threshold is the minimum amount of energy required to consistently cause depolarization and therefore contraction of the heart. Pacing threshold is measured in the terms of both amplitude and duration for which it is applied to the myocardium. The amplitude is programmed in volts (V) or in milliamp in some devices, and the duration is measured in milliseconds. **Table 2** lists the factors affecting pacing threshold.

If pacing threshold of myocardium increases more than that of pacemaker due to any of the above-mentioned reasons, the pacemaker spike will be seen on the ECG but it will not be followed by a QRS complex or cardiac contraction, i.e., failure to capture (**Fig. 4**). This is different from pacemaker failure, where the pacemaker does not fire, and no spike is seen on the ECG (**Fig. 5**).

TABLE 2: Factors affecting pacing threshold.

Factors which increase pacing threshold	Factors which decrease pacing threshold
1–4 weeks after implantation	Increased catecholamines
Myocardial ischemia/infarction	Stress and anxiety
Hypothermia and hypothyroidism	Sympathomimetic drugs
Hyperkalemia and acidosis/alkalosis	Anticholinergics
Antiarrhythmics (classes IA, IB, and IC)	Glucocorticoids
Severe hypoxia/hyperglycemia	Hyperthyroidism
	Hypermetabolic status

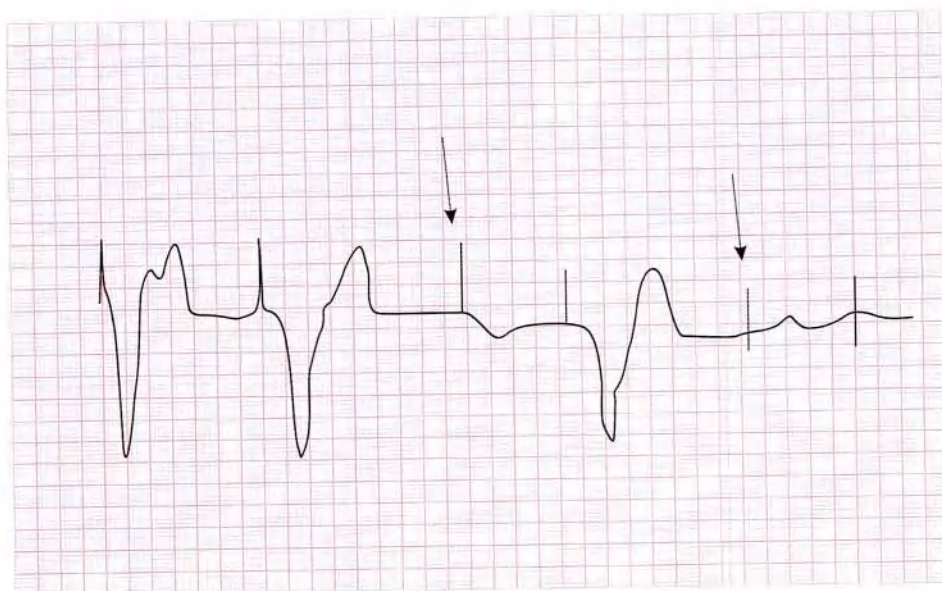


Fig. 4: Failure to capture.

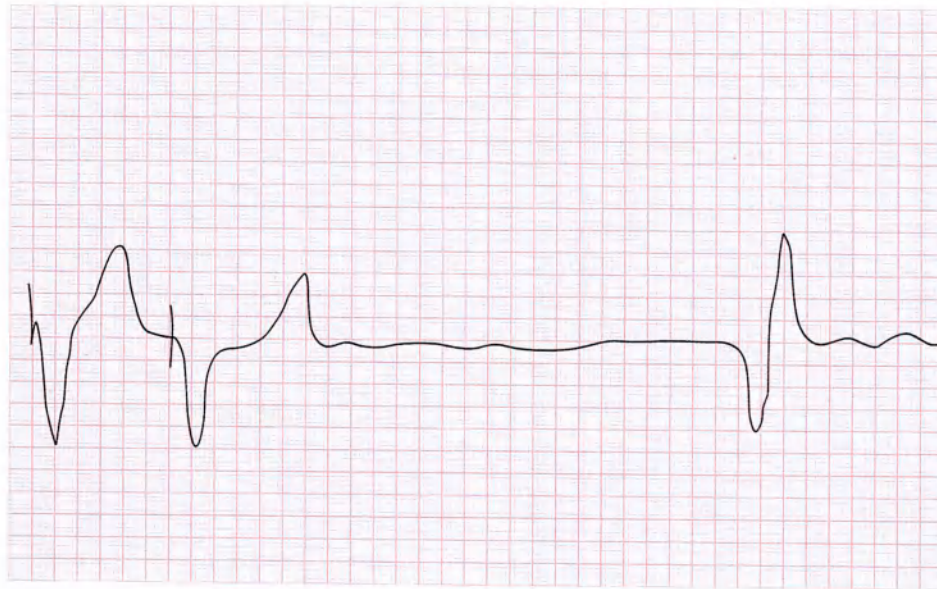


Fig. 5: Failure to pace (absence of spikes).

Q. How can you establish temporary pacing in this case?

Ans. Transcutaneous: Defibrillating electrodes such as Zoll pads are preferred. They can be connected to a defibrillator or pacemaker. The electrodes should be placed as far (>6 inch or 15 cm) from a CRMD. Three recommended electrode placements are as follows:

1. *Anteroposterior placement:* The right arm (RA) electrode placed under the left scapula and the left leg (LL) electrode at apex of the heart.
2. *Posteroanterior placement:* The RA electrode placed under the right clavicle and the LL electrode at the apex of the heart.
3. *Apex posterior placement:* The RA electrode placed over the right scapula and the LL electrode at the apex of the heart.

Transesophageal: Transesophageal atrial pacing is feasible because of the proximity between the esophagus and the posterior aspect of the left atria.

Q. What is transesophageal pacing?

Ans. Transesophageal atrial pacing is feasible in almost all patients because of the proximity between the esophagus and the posterior aspect of the atria. Transesophageal pacing and recording is done using specialized or nonspecialized catheters. There are two different lead types:

1. The pill electrode connected to a flexible wire that the patient swallows with water.
2. A flexible catheter that can even be used in comatose or intubated patients and passed nasally.

It is positioned into the esophagus in order to record the posterior paraseptal atrial electrogram.

There is a relationship between the site of maximal atrial amplitude and the lowest atrial pacing threshold. The optimal atrial pacing site is usually found around 40 cm from the nares.

There are a few limitations to the technique which are as follows:

- There is only one site of atrial pacing and recording.
- There is no ventricular pacing.
- Sometimes, atrial capture can be difficult to assess on the surface ECG.
- Leads to patient discomfort most frequently described as mild burning or chest discomfort such as indigestion.

Q. Discuss intraoperative monitoring for patients with pacemakers.

Ans. Intraoperative monitoring should be based on the patient's underlying disease and the type of surgery. Continuous ECG monitoring is essential to monitor pacemaker functioning. The artifact filter on the ECG monitor should be disabled in order to detect the pacing spikes. In addition, mechanical evidence of the cardiac contraction should be monitored by palpation of the pulse, pulse oximetry, precordial stethoscope, and arterial line, if indicated. Presence of pacemaker is not an indication for insertion of pulmonary artery (PA) or central venous catheter. If these are indicated, care should be taken during insertion of the guidewire or central venous catheter as they are potentially arrhythmogenic and can also dislodge pacemaker leads, especially in recently implanted CRMDs. It is best to avoid the insertion of PA catheter. During the use of unipolar electrocautery, the ECG is frequently unreliable

because of interference. The best monitor to determine if the pacemaker is functioning properly is a hand on the pulse. The precordial or esophageal stethoscope or pulse oximeter can also be used. Since these patients frequently have underlying cardiac dysfunction, other parameters of perfusion should also be monitored. These include capillary refill, urine output, peripheral temperature, etc.

Q. What would be your choice of anesthesia for this patient? What precautions will you take during anesthesia?

Ans. The anesthetic technique should be used according to the need of the patient. Though there are no guidelines favoring or contradicting the use of regional anesthesia in patients with pacemaker, few case reports of use of regional techniques in obstetric are available. One must remember that a paced heart cannot compensate for hypotension by tachycardia and hence spinal anesthesia should be used cautiously and preferably avoided in cases of anticipated blood loss or fluid shift. Since our patient is undergoing total knee replacement (TKR) surgery, femorosciatic block may be given. Spinal anesthesia aimed for block below T12 level or graded epidural analgesia can also be safely used provided that there are no other contraindications for central neuraxial blockade.

Intraoperative management in case of general anesthesia: Both narcotics and inhalational techniques can be used safely. These anesthetic agents do not alter current and voltage thresholds of the pacemaker. Skeletal myopotentials commonly encountered with succinylcholine, myoclonic movements, or direct muscle stimulation can inhibit or trigger pacemaker and should be avoided. Avoid perioperative hypothermia as muscle activity caused by shivering may affect pacemaker functioning. Use of nondepolarizing muscle relaxants is safe. In induction agents, etomidate and ketamine should be avoided as these can sometimes cause myoclonic movements.

Q. Is there a need to reprogram the pacemaker and convert it to asynchronous mode for every surgery?

Ans. Reprogramming the pacemaker to asynchronous fixed mode puts patient at risk of developing R on T phenomenon and VT, especially for patients with good underlying rate. When the risk of EMI is unlikely to interfere with the pacemaker as in surgeries below umbilicus, contralateral arm, on eyes or face or surgeries not requiring use of cautery, the pacemaker need not be reprogrammed. Also for patients who are not dependent on pacemaker as elicited from history and preoperative assessment, it is better to leave pacemaker on demand mode. But in all situations rate responsive function should be suspended.

Q. What are the effects and how will you minimize the effect of electrocautery?

The responses of pacemakers to electrocautery include:

- Inhibition of pacing
- Asynchronous pacing
- Reset to backup mode
- Myocardial burns (rare)
- Ventricular fibrillation (rare).

Responses of ICD include:

- Inhibition of pacing
- Asynchronous pacing
- Inappropriate tachytherapy
- Inhibition of tachytherapy.

One should apply the following measures to decrease the possibility of adverse effects due to electrocautery:

- Bipolar cautery should be used as much as possible as it has less EMI.
- If unipolar cautery is to be used during operation, the grounding plate should be placed close to the operative site (active cautery tip) and as far away as possible from the site of pacemaker, usually on the thigh and should have good skin contact.
- Electrocautery should not be used within 15 cm of pacemaker.
- Frequency of electrocautery should be limited to 1-second bursts in every 10 seconds to prevent repeated asystolic periods. Short bursts with long pauses of cautery are preferred.
- Pacemaker may be programmed to asynchronous mode by a magnet or by a programmer. Before using cautery, the programmer must be available in the operation theater (OT). During the use of cautery, magnet should not be placed on pulse generator as it may cause pacemaker malfunction.
- If defibrillation is required in a patient with pacemaker, paddles should be positioned as far away as possible from the pacemaker generator. If possible, anterior to posterior positioning of paddles should be used. Although permanent pacemakers have protective circuits to guard against externally applied high voltage, pulse generator malfunction has been reported.
- In elective cardioversion, the lowest voltage necessary should be utilized. However, even with these precautions, defibrillation may result in acute increase in the stimulation threshold, with resultant loss of capture. If this occurs, immediate reprogramming or temporary pacing should be done with increased generator output.
- Careful monitoring of pulse, pulse oximetry, and arterial pressure is necessary during electrocautery, as ECG monitoring can also be affected by interference. The device should always be rechecked after operation.

Q. In the middle of surgery, the patient with an ICD develops VT. What would you do?

For a patient with an ICD and magnet-disabled therapies/programming-disabled therapies:

- Avoid all sources of EMI.
- Remove the magnet/reenable therapies through programming, if programmer available.
- Observe patient for appropriate CRMD therapy.

If above activities does not start, the ICD function proceed with external defibrillation/cardioversion. For external cardioversion, place pads or paddles as far as possible from CRMD. Place defibrillator pads perpendicular to the major axis of CRMD to the extent possible by placing them in an anteroposterior location. If technically impossible to place the pads or paddles in location that help to protect the CRMD, defibrillate/cardiovert the patient in the quickest possible way and be prepared to provide pacing through other routes.

Q. Is there a difference between the use of magnet and reprogramming periprocedurally?

- *Use of magnet:*
 - *Advantage:* It can be done easily and fast. It converts to asynchronous mode and deactivates ICD therapy which can be easily reversed by its removal.
 - *Disadvantage:* In varied patient, position placement/maintaining the position of magnet may be difficult.
 - It is important to remember that the magnet must be used by technicians or doctors who are aware of its function and can handle the resultant rhythm.
 - Newer devices may not be magnet sensitive and hence may need reprogramming.
- *Reprogramming of device:*
 - *Advantage:* Concerns of the magnet not correctly placed or accidentally getting dislodged is negated.
 - *Disadvantage:* Needs the special software to communicate and reprogram the device.
 - Takes longer.
 - There remains a possibility that the device may not be reprogrammed at the end of the surgery due to human error.

Q. Are there any special concerns in the postoperative period?

Ans. In patients with rate responsive pacemakers, rate responsive mode should be deactivated before surgery. If this is not possible for some reason, the mode of rate response must be known so that conditions causing changes in paced heart rate can be avoided. For example, shivering and fasciculations should be avoided in the postoperative period. If the pacemaker is “activity” rate responsive—

ventilation should be kept controlled and constant, and temperature must be kept constant in “temperature” rate responsive pacemakers.

Q. In a patient with pacemaker for electroconvulsive therapy (EST), what care would you take?

Ans. Electroconvulsive therapy appears safe for patients with pacemakers, since little current flows within the heart because of the high impedance of body tissue, but the seizure may generate myopotentials which may inhibit the pacemaker. Thus, ECG monitoring is essential and pacemakers should be changed to nonsensing asynchronous mode (fixed mode).

Q. Any others areas where one needs to exercise caution with patient with pacemaker?

Ans. *Transurethral resection of prostate (TURP) and uterine hysteroscopy:* Coagulation current used during TURP procedure has no effect, but the cutting current at high frequencies (up to 2,500 kc/s) can suppress the output of a bipolar demand ventricular pacemaker.

Radiation: Cases where radiation therapy is planned for deep-seated tumors, therapeutic radiation can damage the complementary metal-oxide-semiconductors (CMOSs) that are the parts of most modern pacemakers. Generally, doses in excess of 5,000 rad are required to cause pacemaker malfunction but as little as 1,000 rad may induce pacemaker failure or cause runaway pacemaker. If pacemaker cannot be shielded from the field of radiation, consideration should be given to reimplanting the pacemaker generator at distant site. Temporary damage to pacemaker may recover after reprogramming but there may be permanent damage to the pacemaker as well. This effect could be attributed to charge accumulation inside CMOS after radiation therapy leading to failure of various components in the circuitry and therefore, cause pacemaker failure.

Transcutaneous electronic nerve stimulator (TENS) unit: TENS is now a widely used method for pain relief. TENS unit consists of several electrodes placed on the skin and connected to a pulse generator that applies 20 μ s rectangular pulses of 1–200 V and 0–60 mA at a frequency of 20–110 Hz. This repeated frequency is similar to the normal range of heart rates, so it can create a far field potential that may inhibit a cardiac pacemaker. Adverse interaction between these devices has been frequently reported, so these patients should be monitored during initial application of TENS. Pacemaker-mediated tachycardia has been induced by intraoperative somatosensory evoked potential stimulation.

Studies and case reports suggest that unipolar electrode seems to be most susceptible to interferences.

Nerve stimulators used for regional nerve blocks: One case of pacemaker interference caused by activation of a nerve stimulator has been reported. However, with the advancements of modern pacemaker's technology, a prospective study from Mayo clinic shows that interscalene nerve blocks and other peripheral nerve blocks using the nerve stimulator can be performed in patients with pacemakers without notable interferences with pacemaker functions.

Lithotripsy: Anesthesia may be required in patients undergoing extracorporeal shock wave lithotripsy (ESWL) for immobilization and to avoid pain in flank at entry site of waves. There may be electrical interference from hydraulic shock waves and can cause mechanical damage. High-energy vibrations produced by lithotripsy machine can cause closure of reed switch causing asynchronous pacing. "Activity" rate responsive pacemaker can be affected due to the damage caused to the piezoelectric crystals by ESWL. The shock waves can produce ventricular extrasystoles, if not synchronized with R wave. Pacemaker malfunction can occur in patients undergoing ESWL, requiring adequate preparation prior to procedure. One should have cardiologist's opinion, perioperative ECG monitoring, device programmer, and a standby cardiologist to deal with any device malfunction. Rate responsive pacemaker should have their activity mode deactivated. Focal point of the lithotripter should be kept at least 6 inches (15 cm) away from the pacemaker. Patient with abdominally placed pacemaker generators should not be treated with ESWL.

Dual-chamber demand pacemaker is especially sensitive to shock waves and should be reprogrammed to a simpler mode (VOO and VVI) preoperatively.

Magnetic resonance imaging (MRI): Three types of powerful forces exist in the MRI suite:

1. *Static magnetic field:* An intense static field is always present even if the scanner is not imaging. Most of the pacemakers contain ferromagnetic material, which gets attracted to the static magnetic field in the MRI and may exert a torque effect leading to discomfort at the pacemaker pocket. The reed switch activation by high static field of 0.5–1.5 T can result in switching of pacemaker to a nonsensing asynchronous pacing.
2. *Radiofrequency field (RF):* This field is switched on and off during MRI and has a frequency of 21–64 MHz depending on the strength of magnetic field. The radiofrequency signals can cause interference with pacemaker output circuits resulting in rapid pacing at multiple of frequency between 60 and 300 bpm causing rapid pacing rate. It may cause pacemaker reprogramming and destruction of electronic components. It may also cause heating

at the electrode-tissue boundary, which may cause thermal injury to endocardium and myocardium.

3. *Gradient magnetic field:* Used for spatial localization changes, its strength along different orientations and operates at frequencies in order of 1 kHz. Gradient magnetic field may also interact with reed switch in pacemaker. Inappropriate sensing and triggering because of the induced voltages can occur.

Patients with pacemakers should not routinely undergo MRI scanning. Further studies are necessary to refine the appropriate strategies for performing MRI safely in a patient with implanted pacemaker. The risk-benefit ratio must be individually evaluated in every patient with a pacemaker. Patients who require head MRI scanning without alternative diagnostic possibilities may be best served in a carefully monitored setting. Appropriate patient selection should be done and equipment for resuscitation and temporary pacing should be available. A cardiologist should be present. Also, patients should be closely monitored with ECG and oxygen saturation.

Dental procedures: Detailed history as described is essential. Some important concerns have been addressed:

- *Dental equipment:* Electronic and piezoelectric dental scalers and other dental instruments, except electrosurgery, produce minimal EMI. No cases of adverse events have been reported with the use of these equipment.
- Common dental imaging studies using conventional and digital radiography can be safely done with routine precautions.
- Use of modern chairs with magnets embedded into the cushioning and headrests should be done with caution. It is recommended to consider removing headrests or cushioning and choosing chairs not equipped with magnets.

Q. What is an implantable cardioverter-defibrillator?

Ans. Automatic implantable cardioverter-defibrillator (AICD) system consists of a pulse generator and leads for tachyarrhythmia detection and therapy. It provides antitachycardia and antibradycardia pacing, synchronized or nonsynchronized shocks, telemetry and diagnostics including storing electrograms, and history logs. Modern ICDs use transvenous lead systems for sensing, pacing, and shocks. Epicardial leads are still in use for infants and children. Current ICDs have any programmable features, essentially measure each cardiac R-R interval and categorize the rate as normal, too fast, or too slow. When the device detects a sufficient number of short R-R intervals within a period of time, it will declare a tachycardia episode. The internal computer will decide between antitachycardia

pacing and shock based on its programmed algorithm. Typically, ICD delivers no more than six shocks per episode. Once a shock is delivered, the ICD will redetect to determine whether the shock successfully terminated the arrhythmia (Table 3).

TABLE 3: Types of ICD-NASPE/BPEG generic defibrillator (NBD) code.

Letter I shock chamber(s)	Letter II antitachycardia pacing chamber	Letter III tachycardia detection	Letter IV antibradycardia pacing chamber(s)
O—none	O—none	E—electrogram	O—none
A—atrium	A—atrium	H—hemodynamic	A—atrium
V—ventricle	V—ventricle		V—ventricle
D—dual (A + V)	D—dual (A + V)		D—dual (A + V)

(ICD: implanted cardioverter defibrillator; NASPE: North American Society of Pacing and Electrophysiology; BPEG: British Pacing and Electrophysiology Group).

Indications

- Hemodynamically significant VT or ventricular fibrillation
- Spontaneous sustained VT with structural heart disease
- Syncope of undermined origin with clinically relevant, hemodynamically significant sustained VT or VF induced at electrophysiologic study (EPS).

Q. What are the considerations while giving anesthesia for insertion of a pacemaker?

Ans. A pacemaker is generally inserted under local anesthesia with monitored anesthesia care. Conventionally, cardioverter/defibrillator systems are implanted under general anesthesia. With the development of single-lead transvenous unipolar cardioverter/defibrillator systems for subpectoral implantation a pacemaker-like approach for device implantation appears applicable. Implantation of a single-lead transvenous unipolar cardioverter/defibrillator under local anesthesia with sedation for defibrillation threshold testing is well tolerated by patients.

In case general anesthesia is used in an uncooperative patient, nitrous oxide could cause pacemaker malfunction by increasing gas in the pectoral pacemaker pocket. Despite air evacuation with antibiotic solution before closure of the pectoral pocket, a small amount of air remains entrapped in the pocket. In general, this amount of air should not have clinical significance. However, nitrous oxide is 35 times more soluble in blood than nitrogen. This causes an expansion of the gas in the pocket which leads to loss of a nodal contact and pacing malfunction.

Q. What are leadless pacemakers? Enumerate salient features.

Ans. New devices are now in use which are essentially a self-contained device which is directly delivered into the heart and thus negates the risk of mechanical damage and fractures of leads and also negates any cosmetic concern. These devices are inserted into the right ventricle through the femoral route under fluoroscopic guidance. Once position is acceptable, the device is deployed into the myocardium. Interrogation and reprogramming of the devices can be done through special communication system. Leadless pacing has been proposed in patients with limited venous anatomy and in patients in whom traditional transvenous pacing is contraindicated including superior vena cava obstruction and bilateral subclavian obstruction.

SUGGESTED READING

1. Della Rocca DG, Gianni C, Di Biase L, Natale A, Al-Ahmad A. Leadless pacemakers: state of the art and future perspectives. *Card Electrophysiol Clin.* 2018;10(1):17-29.
2. Ellenbogen KA, Gilligan DM, Wood MA, Morillo C, Barold SS. The pacemaker syndrome—a matter of definition. *Am J Cardiol.* 1997;79(9):1226-9.
3. Fun-Sun FY, Malhotra V, Fontes ML (Eds). Yao and Artusio's *Anesthesiology: Problem-oriented Patient Management*, 6th edition. Philadelphia: Lippincott Williams and Wilkins; 2007. p. 1376.
4. Jacob S, Panaich SS, Maheshwari R, Haddad JW, Padanilam BJ, John SK. Clinical applications of magnets on cardiac rhythm management devices. *Europace.* 2011;13(9):1222-30.
5. Morgan GE, Mikhail MS, Murray MJ (Eds). *Clinical Anesthesiology*. New York: McGraw-Hill, Inc; 2006.
6. Practice advisory for the perioperative management of patients with cardiac rhythm management devices: pacemakers and implantable cardioverter-defibrillators. a report by the American Society of Anesthesiologists Task Force on Perioperative Management of Patients with Cardiac Rhythm Management Devices. *Anesthesiology.* 2011;114:247-61.
7. Rastogi S, Goel S, Tempe D, Virmani S. Anesthetic management of patients with cardiac pacemakers and defibrillators for noncardiac surgery. *Ann Card Anaesth.* 2005;8(1):21-32.
8. Strickberger SA, Conti J, Daoud EG, Havranek E, Mehra MR, Piña IL, et al. Patient selection for cardiac resynchronization therapy: from the Council on Clinical Cardiology Subcommittee on Electrocardiography and Arrhythmias and the Quality of Care and Outcomes Research Interdisciplinary Working Group, in collaboration with the Heart Rhythm Society. *Circulation.* 2005;111(16):2146-50.
9. Tom J. Management of patients with cardiovascular implantable electronic devices in dental, oral, and maxillofacial surgery. *Anesth Prog.* 2016;63(2):95-104.

Peripheral Vascular Disease

Parmanand Jain, Atul Prabhakar Kulkarni, Shilpushp Jagannath Bhosale

A 60-year-old male patient, smoker with painful claudication having gangrene of left foot is scheduled for amputation.

Q. What is peripheral vascular disease?

Ans. Peripheral vascular disease (PVD) is segmental inflammatory vasculitis leading to occlusion of small and medium size arteries and veins in extremities. Thromboangiitis obliterans is a common example. Disease can affect vessels in upper or lower extremities and sometimes that of brain, myocardium, lungs, kidney, and intestine. Patient mostly presents with claudication, rest pain, gangrene, and ulcer. Raynaud's phenomenon may accompany the disease. Superficial thrombophlebitis may lead to pain and edema. Ischemic pain is more during night and is aggravated by elevation of limb. There may be paresthesias due to nerve involvement. The disease progress is slow and symptoms are intermittent. PVD is a powerful predictor of future cerebrovascular and cardiovascular events such as myocardial infarction (MI) and stroke, and of increased mortality.

Q. What is the difference between Buerger's disease and thromboangiitis obliterans?

Ans. Difference between Buerger's disease and thromboangiitis obliterans is shown in **Table 1**.

TABLE 1: Difference between Buerger's disease and thromboangiitis obliterans.

Buerger's disease	Thromboangiitis obliterans
Age: 20–45	Age: 45 and above
Mostly males	Mostly males. Female 10%
Upper limbs	Upper limbs seldom involved
Superficial veins	Deep veins occasionally
Onset ~ foot pain, claudication	Onset ~ calf claudication
Gangrene often early, nearby pulses present	Gangrene 10%, later, loss of pulses
Smoking +	Smoking +, other factors

Contd...

Contd...

Buerger's disease	Thromboangiitis obliterans
Sympathectomy/anticoagulants	Sympathectomy and anticoagulation doubtful
Cholesterol normal	Cholesterol high
No calcification	Calcification present
Normal electrocardiogram (ECG)	Diabetes mellitus
Life prognosis good	Life prognosis reduced

Q. What is Takayasu's arteritis?

Ans. Takayasu's arteritis is an idiopathic, chronic, progressive occlusive vasculitis that causes narrowing, thrombosis, or aneurysms of systemic and pulmonary arteries. Inflammatory changes are seen preferentially in large blood vessels such as the aorta and its branches.

Q. What is Raynaud's phenomenon? Why does it occur?

Ans. It is episodic vasospastic ischemia of the digits. It is seen more often in women. It is characterized by digital blanching, cyanosis, and rubor after cold exposure and rewarming. Blanching represents the ischemic phase of the phenomenon caused by digital vasospasm.

Q. Define claudication.

Ans. Claudication can be described as functional ischemia characterized by reproducible lower extremity muscle pain induced by a defined amount of activity. The pain is relieved by rest. The symptoms are due to the inability of the vasculature to provide blood flow according to the metabolic demand of the exercising tissue.

Claudication at rest is described as critical ischemia where arterial perfusion is inadequate to meet the resting demands of tissue and is indication for surgical intervention.

Up to 15% of patients with asymptomatic peripheral arterial disease (PAD) will develop symptoms of intermittent

claudication or critical limb ischemia and a further 1–3% of these patients will require major limb amputation within a 5-year period.

Peripheral arterial disease most commonly affects the superficial femoral artery giving rise to calf claudication pain. Occlusion of the aortoiliac, common femoral, and tibio-peroneal vessels gives rise to pain in the buttock, thigh, or foot, respectively. Symptoms are often insidious, developing over months to years with a gradual reduction in pain-free walking distance. Ischemic rest pain and tissue loss (i.e., ulcers or gangrene) are key signs of critical limb ischemia and will require either endovascular intervention or peripheral vascular revascularization (PVR) surgery.

Q. What are preanesthetic considerations in PVD?

Ans. The American College of Cardiology and the American Heart Association (ACC/AHA) guidelines on perioperative management classify PVR as high-risk surgery with a combined cardiac death and nonfatal MI of >5%.

Peripheral vascular disease is commonly seen in elderly and males are more likely to develop PAD compared with females with a male to female ratio of 2:1.

Baseline evaluation of vital signs including bilateral blood pressures, heart rate, height, and weight should be obtained. Systolic blood pressures ideally should be within 10–15 mm Hg in both limbs. Systemic evaluation mainly cardiac and pulmonary examinations should be performed to assess cardiac manifestations of atherosclerosis, arrhythmias responsible for thromboembolism, and operative risks.

Respiratory: Preoperative evaluation includes history of smoking and chronic obstructive pulmonary disease (COPD). Smoking is directly related to the development of PAD with the amount and duration of tobacco use being directly correlated to the progression of PAD. Symptomatic PAD presents ~10 years earlier in smokers than in nonsmokers, thus making smoking a significant modifiable risk factor.

Pulmonary function test (PFT) helps to determine whether regional versus general anesthesia (GA) is appropriate, while providing baseline values for postoperative comparison.

Tests: Chest X-ray (CXR), consider PFTs and arterial blood gas (ABG).

Cardiovascular: There is a higher prevalence of coronary artery disease (CAD) with up to 60% of vascular surgery patients having severe CAD. Patients with symptomatic PAD have a four- to sevenfold increase from all-cause mortality and a 15-fold increase in cardiovascular-related mortality compared with those without PAD at 10 years. There is also a higher incidence of renal disease, cerebral vascular disease, and diabetes in vascular patients.

Tests: Electrocardiogram (ECG) and two-dimensional (2D) echocardiography, dipyridamole thallium imaging (DTI), radionuclide ventriculography and dobutamine stress echocardiography.

Neurological: Patients with PVD have increased incidence of cerebrovascular disease. Careful neurological assessment is necessary to document existing deficits.

Endocrine: For patients with diabetes, the hemoglobin A1c (HbA1c) level is strongly associated with the risk of developing PAD with a 1% increase in HbA1c being associated with a 26% increase in the risk of PAD, and patients with a HbA1c > 7.5% are five times more likely to have symptomatic PAD.

Increase incidence of diabetes mellitus may be associated with peripheral and autonomic neuropathies (silent MI, labile blood pressure) and delayed gastric emptying. Frequent blood glucose measurements should be made preoperatively.

Renal: There is a higher incidence of renal artery disease and renal insufficiency in this patient population.

Test: Blood urea nitrogen (BUN) and creatinine; consider creatinine clearance; electrolytes and uric acid.

Hematologic: Many patients presenting for this surgery are taking anticoagulant/antiplatelet medication. Inquire as to bleeding or bruising tendency.

High levels of low-density lipoprotein (LDL) cholesterol and low levels of high-density lipoprotein (HDL) cholesterol increase the likelihood of developing PAD. Cardiac-related mortality is reduced by 20% for every 1 mmol/L reduction in LDL cholesterol. High homocysteine level is also an independent risk factor for PAD with 30% of patients with PAD having an elevated homocysteine level compared with just 1% of the general population.

Tests: Hematocrit, platelet count; prothrombin time (PT) and partial prothrombin time (PTT), and lipid profile.

Q. How do you investigate a case of PVD?

Ans. The PVD patients commonly come for emergency surgeries and optimum workup may not be always possible due to the associated comorbidities. Patients with PVD are at high risk for developing postoperative complications and the long-term survival in such patients is <65%.

Aim of preanesthetic evaluation is:

- To rule out other causes of vascular obstruction like atherosclerosis, collagen vasculitis and hypercoagulability.
- To exclude proximal source of emboli and involvement of larger vessels.
- To find out organ involvement, to find out systemic effects due to smoking.

Preoperative Risk Assessment

The Lee's Revised Cardiac Risk Index (RCRI) is an assessment of cardiac risk in noncardiac surgery patients and has been extensively validated and used in vascular surgical patients to predict cardiac outcome. The RCRI consists of six cardiac risk factors and the presence of three or more risk factors is predictive of a minimum 11% risk of major perioperative cardiac event.

The increase in morbidity and mortality in patients undergoing PVR is mainly attributed to preexisting CAD. Most patients with symptomatic PAD have significantly impaired mobility due to claudication pain, which limits their ability to perform daily activities and hence cardiorespiratory symptoms are masked. A thorough clinical assessment is necessary to elicit cardiorespiratory symptoms, functional capacity, and modifiable risk factors. Functional capacity is assessed subjectively based on the patient's exercise tolerance and can be expressed as metabolic equivalents (METs). Poor exercise tolerance (<4 METs) is associated with an increase in perioperative myocardial ischemia and cardiac events. Patients with one or more major cardiac risk factors as per the ACC/AHA guidelines should have their procedure deferred for more extensive cardiac investigations and management unless the procedure is emergent. Patients presenting for elective PVR with METs < 4, dyspnea of unknown origin, poorly controlled cardiac failure, or cardiac risk factors should be considered for thorough noninvasive cardiac evaluation.

Nonexercise stress testing is preferable for patients with PAD and critical leg ischemia (CLI) for cardiac evaluation. Stress echocardiogram has a high sensitivity for predicting cardiac ischemia but has an overall low positive predictive value for perioperative cardiac events. Its main value is in its high negative predictive value ranging from 93 to 100% for MI and cardiac death. Cardiopulmonary exercise testing and exercise ECG testing are of limited value in patients with symptomatic PAD due to limitations in their mobility. Cardiac biomarkers such as B-type natriuretic peptide (BNP) and N-terminal pro-BNP (NT-proBNP) have been shown to be independently predictive of left ventricular (LV) failure, MI, and 30-day and longer-term cardiac mortality.

Q. How will you investigate a patient with PVD?

Ans.

- **Complete blood count (CBC):** Polycythemia, infection, erythrocyte sedimentation rate (ESR), blood grouping and crossmatching.
- **Urine:** Albumin, sugar, blood sugar—serum creatinine, liver function test (LFT).
- **Lipid profile:** Triglycerides, serum cholesterol, HDL and LDL.
- Antinuclear antibody (ANA), rheumatoid factor, antiendothelial cell antibodies.
- **Prothrombotic states:** Decreased antithrombin protein C, protein S, prothrombin.
- Antiphospholipid antibodies, complement, fibrinogen.
- ECG, dobutamine stress echocardiography.
- **X-ray chest:** COPD and calcified aorta.
- **ABG:** PCO₂, PaO₂: Baseline.
- **Doppler ultrasound:** Blood flow detection (ABPI).
- An efficient method of objectively documenting the presence and severity of lower extremity PVD is determination of the ankle-brachial pressure index (ABPI). This is done by measuring blood pressure at the ankle and in the arm while a person is at rest. Measurements are usually repeated at both sites after 5 minutes of walking on a treadmill. The ratio of ankle systolic pressure to brachial artery systolic pressure is called ABPI. During exercise, the increased flow through the narrowed vessels leads to decrease in pressure causing fall in ankle pressures. An ABPI between 1 and 1.2 is considered normal, 0.3–0.9 indicates arterial disease and < 0.3 indicates impending gangrene. When compared to angiography, the sensitivity of the ankle-brachial index (ABI) is about 90%, and the specificity is about 98% for stenosis of 50% or more in leg arteries. The ABI is not reliable in the setting of calcified vessels that become either non- or poorly compressible, most commonly seen in patients with diabetes mellitus or renal failure. The toe-brachial index is usually a more reliable indicator of obstructive PAD in this patient population. The toe-brachial index is performed by placing a small occlusion cuff on the great toe with a photoplethysmography sensor on the tip of the toe.
- **Pulse volume recording:** It measures the volume and flow pattern into a limb. A cuff is inflated to a known pressure of 60 mm Hg and kept constant. Arterial inflow displaces the cuff air, generating an increase in pressure that is recorded over time. A normal pulse volume recording has a rapid upstroke to a peak in systole, with a dicrotic notch and subsequent descent during diastole. When arterial stenosis is present, the upstroke becomes slurred, the peak becomes flattened, and the dicrotic notch is lost. The advantage of pulse volume recording is that, unlike the ABI, it is independent of arterial calcification.
- Continuous-wave Doppler is most often used as part of the ABI test. The probe is placed over the artery to be measured, and the loss of and return of flow in the vessel is recorded. The qualitative analysis of a normal triphasic signal can be recorded. A normal continuous-wave Doppler is triphasic, with a rapid upstroke or forward flow, a downstroke to below baseline as flow reverses,

and finally a short period of forward flow is seen again. When stenosis is present and as it increases, the reversal of flow is lost and the upstroke may be delayed. With greater stenosis, the upstroke becomes smaller and further delayed (*parvus et tardus*), sinusoidal, and eventually absent.

- Duplex imaging can show image of vessel, velocity of blood flow as good as arteriography.
- *Isotope scanning*: Perfusion of limb.
- Digital subtraction angiography.
- *Computed tomography (CT)/magnetic resonance (MR) angiography*: Corkscrew or root-tree appearance due to collaterals.

Q. What are the factors, which predict perioperative morbidity and mortality?

Ans. The ACC/AHA clinical predictors of perioperative risks:

- Major:
 - Unstable angina/MI in 30 days
 - Congestive cardiac failure (CCF)
 - Arrhythmias
 - Severe valvular disease
- Intermediate:
 - Mild angina
 - MI (30 days earlier)
 - Compensated CCF
 - Diabetes mellitus
 - Renal insufficiency
- Minor:
 - Age > 70 years
 - Abnormal ECG
 - Rhythm other than sinus
 - History of stroke
 - Uncontrolled hypertension.

Q. What is medical treatment of PVD?

Ans.

- Control hypertension, CAD, diabetes mellitus, dyslipidemia, hypothyroidism, atherosclerosis, bronchospasm, respiratory infection.
- Stop smoking, protection from trauma, infection and cold.
- *Analgesics*: Nonsteroidal anti-inflammatory drugs (NSAIDs), narcotics.
- Warfarin, antiplatelet (aspirin, clopidogrel), angiotensin-converting enzyme (ACE) inhibitors, pentoxifylline.
- Statins decrease the incidence of intermittent claudication.
- *Cilostazol*: 100 mg BD (phosphodiesterase inhibitor) increase exercise time to development of intermittent claudication.

- Prostaglandin E1 and E2 infusions.
- Intra-arterial urokinase.
- Omentopexy, subtotal adrenalectomy.
- Angioplasty, excisional atherectomy, bypass surgery, and even amputation.

All patients with PVD should be on antiplatelet drugs and clopidogrel has been shown to be more effective than aspirin. Evidence suggests clopidogrel to be superior to aspirin with a 23.8% relative risk reduction for cardiovascular complications in patients with PVD. Discontinuation of clopidogrel and the newer antiplatelet agents (e.g., ticagrelor, prasugrel) perioperatively should be decided on an individual patient basis taking into account the indication for the drug and the risks and benefits of acute cessation. Aspirin is considered safe to be continued throughout the perioperative period. Warfarin should be stopped before surgery with a target international normalized ratio of <1.5 for neuroaxial anesthesia and <2.0 for GA considered to be safe. Treatment of hypertension is indicated to aim for arterial pressure <140/90 in patients with PVD and <130/80 in patients with PVD and diabetes to reduce the risk of cardiovascular events. ACE inhibitors have been shown to significantly reduce the risk of fatal and nonfatal ischemic events in patients with PVD even if they are normotensive and asymptomatic. Statins should be commenced for all patients with established PVD and continued after operation.

Q. What are indications for surgery in PVD (lower extremity percutaneous transluminal angioplasty or bypass surgery)?

Ans.

- Incapacitating claudication in persons; interfering with work or lifestyle.
- Limb salvage in persons with limb-threatening ischemia as manifested by rest pain, nonhealing ulcers, and/or infection or gangrene.
- Vasculogenic impotence.

Q. What are anesthetic implications of PVD surgery?

Ans. Anesthesia is generally required for:

- Distal bypass procedures (infrainguinal artery bypass, lumbar sympathectomy, venous thrombectomy or vein excision), revascularization [tibioperoneal/saphenous vein graft or polytetrafluoroethylene (PTFE)], amputation.
- Diagnostic and therapeutic sympathetic block.
- Radiofrequency thermocoagulation of sympathetic chain.
- Spinal cord stimulation/implantation, treatment of postamputation pain and phantom limb pain.

Q. How do you give anesthesia in PVD? How do you monitor intraoperatively?

Ans.

- Anesthesia for the acutely ischemic lower limb is challenging because of inadequate time to evaluate and treat comorbidities and the physiological disturbances from an ischemic limb.
 - Local anesthesia with invasive monitoring by an anesthesiologist is the preferred technique for embolectomy.
 - Revascularization may still require GA.
 - Regional anesthesia can reduce postoperative thrombotic events, minimize cardiac morbidity and maintain effective postoperative analgesia.
 - Peripheral nerve blocks are utilized in special situations with minimal interference with cardiovascular function.
 - For infrainguinal arterial bypass, there is evidence that regional anesthesia is superior for promoting graft survival.
 - For GA gradual induction (small increments of fentanyl 1–2 µg/kg and thiopentone 50–75 mg until asleep) is desirable. If LV function is poor, high dose fentanyl induction is preferred.
 - Use warm fluids and humidified gases to prevent hypothermia.
 - Essential to use perioperative fluid regimen, which reduce blood viscosity and enhance blood flow. Colloid-containing fluids are preferred to crystalloids owing to better intravascular volume supporting capacity and specific colloid-associated pharmacological effects on rheological variables.
- Irrespective of anesthetic technique, good postoperative outcome is more likely to be influenced by the meticulous management of oxygenation, hemodynamic stability, fluids, temperature, and analgesia to minimize perioperative complications. Tachycardia should be avoided perioperatively as it increases myocardial oxygen demand while decreasing myocardial perfusion, which can result in myocardial ischemia and infarction. Large variations in arterial pressure, even for short durations, can be equally detrimental by increasing the risk of myocardial ischemia, strokes, renal dysfunction, postoperative cognitive dysfunction (POCD), and surgical bleeding.

Monitoring:

- ST-segment analysis.
- Invasive arterial pressure monitoring is indicated for high-risk patients and for prolonged procedures. Cardiac output monitoring can be useful to guide fluid management and monitor LV function in patients at higher risk of perioperative cardiac complications.

- Be careful in patient positioning and pad pressure points, eyes. Diabetic patients may be at risk of skin ischemia due to poor positioning or inadequate padding of limbs, etc.

Q. Does regional blockade alter outcome?

Ans. Regional anesthesia does have several advantages over GA that it modifies the cardiovascular and metabolic responses to the stress of surgery and helps to diminish myocardial ischemic episodes. The sympathetic blockade and vasodilatation produced improves the graft perfusion and decreases the chances of graft failure. The circulating levels of catecholamines are also reduced, which decrease the probability of vasoconstriction. There is increased fibrinolysis too due to decrease in procoagulant factors. All these factors put together definitely improve the graft survival. Regional anesthesia also reduces respiratory morbidity and POCD while providing good quality postoperative analgesia.

Regional anesthesia is contraindicated in patients who are coagulopathic and the risks *versus* benefits of regional anesthesia must be carefully considered for patients on dual antiplatelet therapy.

Q. What is the role of lumbar sympathetic block in PVD? How do you perform the block?

Ans.

- *Anatomy:* Sympathetic contribution to lower extremity arises from cell bodies originating in lateral horn of spinal cord (T10–L2). This segment of sympathetic chain runs along L2, L3, L4 vertebral bodies.
- *Contraindications:* Coagulopathy, infection.
- *Position:* Lateral or prone.
- *Diagnostic block/therapeutic block:* 10 mL 0.25% bupivacaine is injected at two sites, with 22-gauge needle of 15 cm length.
- *Sympathetic block success:* Temperature rise of >20°C on ipsilateral leg, improvement in pain, motor function and color usually occurs within minutes establishing diagnosis.
- No reliable evidence to support its use in Buerger's disease, intermittent claudication, diabetic vascular disease or ischemic ulceration or gangrene.

Q. What are the postoperative complications in PVD surgery?

Ans.

- Hypertension/hypotension, hypothermia, tachycardia, hypoxemic episodes, hypercapnia, myocardial ischemia/infarction and renal functional disturbances, congestive heart failure (CHF), hypothermia.
- Graft occlusion; hypothermia causes vasoconstriction and also may limit outflow to the graft.

- Avoid overhydration; when epidural sympathectomy fades; increased risk for CHF.
- Maintain normovolemia; so that peripheral vasoconstriction does not occur.

SUMMARY

Patients with PVD are generally associated with CAD and/or carotid artery disease. Though exercise tolerance is a useful indicator of the severity of CAD, it is almost always limited by intermittent claudication or other comorbidities. Vascular reconstructions are of prolonged duration and are associated with hypovolemia and hypothermia. Myocardial ischemia is very common and often associated with infarction.

SUGGESTED READING

1. Bode RH Jr, Lewis KP, Zarich SW, Pierce ET, Roberts M, Kowalchuk GJ, et al. Cardiac outcome after peripheral vascular surgery. Comparison of general and regional anesthesia. *Anesthesiology*. 1996;84:3-13.
2. Christopherson R, Beattie C, Frank SM, Norris EJ, Meinert CL, Gottlieb SO, et al. Perioperative morbidity in patients randomized to epidural or general anesthesia for lower extremity vascular surgery. *Perioperative Ischemia Randomized Anesthesia Trial Study Group*. *Anesthesiology*. 1993;79:422-34.
3. Diehm C, Schuster A, Allenberg JR, Darius H, Haberl R, Lange S, et al. High prevalence of peripheral arterial disease and co-morbidity in 6880 primary care patients: cross-sectional study. *Atherosclerosis*. 2004;172:95-105.
4. Naesh O, Haljamäe H, Hindberg I, Holm J, Jivegård L, Wennmalm Å, et al. Epidural anaesthesia prolonged into the postoperative period prevents stress response and platelet hyperaggregability after peripheral vascular surgery. *Eur J Vasc Surg*. 1994;8:395-400.
5. Rooke TW, Hirsch AT, Misra S, Sidawy AN, Beckman JA, Findeiss LK, et al. 2011 ACCF/AHA Focused update of the guideline for the management of patients with peripheral artery disease (updating the 2005 guideline): a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol*. 2011;58:2020-45.
6. Rosenfeld BA, Beattie C, Christopherson R, Norris EJ, Frank SM, Breslow MJ, et al. The effects of different anesthetic regimens on fibrinolysis and the development of postoperative arterial thrombosis. *Perioperative Ischemia Randomized Anesthesia Trial Study Group*. *Anesthesiology*. 1993;79:435-43.
7. Tovey G, Thompson JP. Anaesthesia for lower limb revascularization. *Contin Educ Anaesth Crit Care Pain*. 2005;5:89-92.

Pneumonectomy

Priya Ranganathan, Madhavi Shetmahajan, Bhakti Trivedi

A 55-year-old male patient presents with history of cough with mucoid expectoration and progressive weight loss since 2 years. After bronchoscopy, computed tomography (CT) scan, and biopsy, he is diagnosed to have bronchogenic carcinoma involving the left lower bronchus, extending into the left upper and left main bronchus. The patient has been smoking 15–20 cigarettes per day for the last 30 years. He gives a history of breathlessness since 5–6 years for which he is taking Salmeterol inhaler twice daily. He has a limited effort tolerance and cannot climb more than one flight of stairs without becoming breathless. There is no other medical history. On examination, his height is 158 cm and weight is 48 kg. He has decreased air entry over the entire left hemithorax with bronchial breath sounds and scattered wheeze. Blood investigations are normal. Chest X-ray shows left middle and lower zone opacity with left mediastinal shift. Electrocardiogram (ECG) shows left bundle branch block (LBBB). Echocardiography shows normal biventricular function with no evidence of pulmonary hypertension. The patient is posted for a left pneumonectomy.

Q. Describe the preanesthetic evaluation of a patient posted for lung resection surgery.

Ans. Preanesthetic evaluation should include:

- A detailed medical history for any coexisting disease. Optimal treatment and control of associated medical conditions should be achieved.
- The patient's functional capacity should be assessed.
- Since patients with lung cancer are usually smokers, a history of smoking and of symptoms suggestive of chronic obstructive pulmonary disease (COPD) should be elicited.
- The patients should be evaluated for ischemic heart disease since they tend to be chronic smokers, which predispose them to atherosclerosis. Patients with major factors for increased perioperative cardiovascular risk should undergo a preoperative cardiologic evaluation.
- Airway evaluation should be performed keeping in mind that these patients are candidates for lung isolation intraoperatively.
- Patients may receive chemotherapy preoperatively and should be evaluated for chemotherapy-related toxicity.
- Investigations should include complete blood count (CBC). This may show polycythemia due to COPD or leukocytosis, which may indicate active pulmonary infection. Sputum cultures and sensitivity studies can be used to guide appropriate antibiotic therapy. Liver and renal functions should be assessed in view of age.
- The chest X-ray (CXR) should be evaluated for tracheal deviation or obstruction, which could predict difficulty with intubation or ventilation, mediastinal mass, which could lead to difficulty with ventilation or superior vena cava (SVC) syndrome, pleural effusions, or areas of consolidation.
- Pulmonary function tests (PFTs) should be carried out to diagnose obstructive or restrictive abnormalities, to assess responsiveness to bronchodilators, and to confirm suitability for resection.
- The electrocardiogram (ECG) should be evaluated for signs of left or right heart dysfunction.
- Transthoracic echocardiography should be carried out to rule out pulmonary hypertension.
- Further cardiopulmonary testing may be indicated based on the results of investigations.

Q. Why is it important to assess suitability for lung resection?

Ans. Lung resections are associated with a high risk of morbidity and mortality. Smoking leads to abnormal pulmonary function and predisposes to both immediate perioperative complications and long-term disability following surgical resection. Loss of lung parenchyma can lead to hypoxemia. In addition, the entire pulmonary blood flow is delivered to the remaining lung, leading to pulmonary hypertension and right heart strain. This can lead

to right ventricular failure. Patients need to be evaluated to see if they can withstand the loss of the resected lung without becoming pulmonary cripples.

In addition, the following points need to be kept in mind:

- Resections may be more extensive than planned.
- Handling of the lung/trickling of blood or secretions may cause postoperative dysfunction of the remaining "normal" lung.

Q. What are the four Ms, which need to be assessed in patients with lung cancer?

Ans. The four Ms, which need to be assessed in patients with lung cancer, are:

- **Mass effects:** Pneumonia, SVC syndrome, nerve palsy
- **Metabolic effects:** Eaton-Lambert syndrome, hypercalcemia
- **Metastases:** To distant organs—bone, adrenals
- **Medications:** Effects of chemotherapy and radiation therapy.

Q. Describe the preoperative respiratory assessment of this patient to assess suitability for pneumonectomy.

Ans. Three aspects of respiratory function need to be assessed preoperatively. These include:

- Lung mechanics
- Lung parenchymal function
- Cardiopulmonary interaction.

Q. How will you assess lung function at the bedside?

Ans. Refer to chapter on Anesthetic Management of a Patient with COPD.

Q. How is adequacy of lung mechanical function assessed?

Ans. The assessment of lung mechanics includes spirometry and determination of lung volumes. Earlier, absolute values of pulmonary functions were used to determine suitability for lung resection.

However, a more valid method of assessing suitability for lung resection is calculation of the predicted postoperative FEV₁ (forced expiratory volume) and DLCO (diffusing capacity of the lung for carbon monoxide).

Q. How is postoperative lung function after lung resection calculated?

Ans. The lungs contain 42 subsegments as shown in Figure 1.

Predicted postoperative FEV₁ (ppoFEV₁%) is calculated by counting the number of segments to be removed:

$$\text{PpoFEV}_1\% = \text{Preoperative FEV}_1\% \times [1 - (\text{No. of subsegments removed}/42)]$$

For example, in a right upper lobectomy, six segments are resected:

Therefore,

$$\text{ppoFEV}_1\% = \text{Preoperative FEV}_1\% \times [1 - (6/42)] = \text{PreopFEV}_1 \times 36/42$$

Patients with a ppoFEV₁ < 40% of the predicted normal value (calculated on the basis of age, height, gender, and race) for that patient are at high risk for postoperative pulmonary complications. Some centers accept ppo functions as low as 30% predicted.

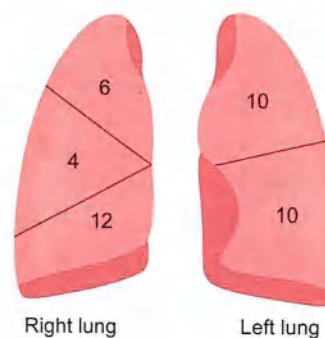


Fig. 1: Subsegments of lungs.

Q. How is lung parenchymal function assessed?

Ans. Conventionally, preoperative arterial blood gas (ABG) values were used to assess patients undergoing lung resection, with PaCO₂ > 45 mm Hg, PaO₂ < 60 mm Hg, and SaO₂ < 90% being considered as risk factors. However, the DLCO has been shown to be an independent predictor of poor outcome after lung resection and is the most useful test of gas exchange.

Predicted postoperative DLCO values (ppoDLCO%) can be calculated using the same formula as for ppoFEV₁. PpoDLCO < 40% of the predicted normal value is a predictor of increased respiratory and cardiac complications.

Q. What are the cutoff values for predicted postoperative lung function?

Ans. Conventionally used cutoff values suggest that ppoFEV₁ < 1 L results in sputum retention and ppoFEV₁ < 800 mL can leave the patient ventilator-dependent. A more accepted estimate of the predicted postoperative lung function is a ppoFEV₁ < 40% or ppoDLCO < 40% indicating a high risk of perioperative complications including death. PpoFEV₁ and ppoDLCO are independent of each other and should be measured in all patients undergoing lung resection. Some studies suggest that patients with ppoFEV₁ and/or ppoDLCO values as low as 30% may also be considered for surgery. Another index, which is used for risk stratification, is the predicted postoperative product (PPP), which is a product of ppoFEV₁% and ppoDLCO%. A PPP < 1,650 has been shown to predict postoperative complications; however, further studies are needed to validate this.

Q. Why is cardiopulmonary exercise testing important in these patients?

Ans. Patients presenting for lung resection usually have a long-standing history of smoking. This predisposes them to atherosclerosis, ischemic heart disease, and COPD. This in turn may lead to left and right ventricular dysfunction. These patients, therefore, may be at higher risk of perioperative myocardial ischemia. In addition, the pulmonary vasculature becomes rigid due to chronic lung disease. Further reduction in the pulmonary vascular bed after lung resection puts the patient at risk for postoperative increases in pulmonary vascular pressure and right heart failure.

Q. What cardiovascular workup is needed?

Ans. Formal exercise testing where maximal oxygen consumption ($\text{VO}_{2\text{max}}$) can be measured is the gold standard for evaluating the cardiopulmonary interaction. $\text{VO}_{2\text{max}} > 15 \text{ mL/kg/min}$ is considered acceptable and these patients are expected to tolerate pneumonectomy, $< 10 \text{ mL/kg/min}$ deems the patient inoperable, and those with $10\text{--}15 \text{ mL/kg/min}$ are considered high risk and require careful evaluation before surgery. However, formal exercise stress testing is expensive and time-consuming and maybe indicated in patients with exercise limitation without a clear cardiac or respiratory cause.

Several alternatives have been developed as substitutes to exercise testing for prethoracotomy assessment.

Stair climbing: The patient is asked to climb stairs at his own pace without stopping. A flight of stairs is taken as 20 steps at 6 inches/step. Patients who can climb five flights of stairs have a $\text{VO}_{2\text{max}}$ over 20 mL/kg/min ; those who manage less than one flight equate to $< 10 \text{ mL/kg/min}$. The ability to climb fewer than two flights is considered high risk.

Shuttle walk test: Patients walk between two markers, 10 meters apart. Their speed is gradually increased every minute. The inability to complete 25 shuttles suggests a $\text{VO}_{2\text{max}} < 15 \text{ mL/kg/min}$.

6-minute walk test: The patient is made to walk on level ground at the maximum pace possible without discomfort. The distance covered and oxygen saturation are monitored. A 6-minute walk test distance of $< 2,000$ feet correlates with a $\text{VO}_{2\text{max}} < 15 \text{ mL/kg/min}$. Desaturation of $> 4\%$ from the resting value during the test is associated with an increased risk of postoperative complications. Dyspnea and fatigue scores are also assessed.

This patient's pulmonary functions are as follows:

$\text{FEV}_1 = 1.04 \text{ L}$ (51% predicted)
[6% change after bronchodilator]

Forced vital capacity (FVC) = 1.21 L (55% predicted)

[5% change after bronchodilator]

$\text{FEV}_1/\text{FVC} = 87\%$ predicted

Maximal voluntary ventilation (MVV) = 54% predicted

[8% change after bronchodilator]

DLCO = 50% predicted

Arterial blood gas on room air shows PO_2 of 83 mm Hg, PCO_2 of 37 mm Hg, and saturation of 97% with normal pH.

Q. Interpret these PFTs and calculate ppoFEV₁ and ppoDLCO after left pneumonectomy. Is this patient suitable for a left pneumonectomy?

Ans. Table 1 describes the changes in PFT in obstructive, restrictive, and mixed defects.

This patient's PFTs show moderate restriction with no obstruction and no reversibility with bronchodilator. There is a decrease in diffusion capacity. ABG shows mild hypoxemia with no CO_2 retention.

TABLE 1: Changes in pulmonary function tests.

	FVC	FEV_1/FVC
Obstructive	Normal	Low
Restrictive	Low	Normal
Mixed	Low	Low

(FEV: forced expiratory volume; FVC: forced vital capacity).

- After left pneumonectomy, the patient will be left with 22 out of 42 lung subsegments.
- $\text{PpoFEV}_1\%$ after left pneumonectomy = $51\% \times 22/42 = 26\%$ which is less than the cutoff of 40%.
- $\text{PpoDLCO}\%$ after left pneumonectomy = $50\% \times 22/42 = 26\%$ which is less than the cutoff of 40%.

Based on these predicted postoperative values, the patient appears unfit for a left pneumonectomy.

Q. What are the limitations and alternatives to this method of calculating postoperative lung function?

Ans. Calculation of ppo lung functions using the above formula assumes that the entire lung is uniformly contributing to ventilation and perfusion. However, the diseased lung may be nonfunctional with very little ventilation and/or blood flow. Thus, resection of that part will not impact the lung function. When any of the whole-lung pulmonary function values are worse than the cutoff limits, the function of each part of the lung needs to be assessed separately. This consists of measurement of the ventilation and perfusion (V/Q) of each lung using radioactive isotope scanning. V/Q scanning allows detailed assessment of the functional capacity of the lung and accurate determination of which lobes or segments contribute proportionally to gas exchange before their resection.

However, the V/Q scan reports have to be read carefully to see whether the reporting is done on the basis of anatomical segments or nonanatomical zones. Zones do not correspond accurately to the lobes of the lung; therefore, values of zonal perfusion/ventilation may not accurately reflect the perfusion/ventilation of lobes when used for calculation of predicted postoperative functions.

Q. A perfusion scan shows 13% perfusion to the left lung and 87% perfusion to the right lung. Based on these findings, is the patient suitable for a left pneumonectomy?

Ans. This patient's FEV₁ is 51%. The right lung has 87% perfusion.

The ppoFEV₁% after left pneumonectomy is $87\% \times 51 = 44\%$.

Similarly, ppoDLCO% after left pneumonectomy is $87\% \times 50 = 44\%$. Both these values are above the cutoff of 40%; therefore, this patient appears suitable for a left pneumonectomy.

Q. What further tests can be done?

Ans. If the second-phase criteria are not met, then tests are conducted to mimic postoperative conditions by temporary occlusion of the corresponding main stem bronchus or pulmonary artery. A rise in pulmonary artery pressure > 45 mm Hg, PaCO₂ > 60 mm Hg, or a fall in PaO₂ < 45 mm Hg indicates inability to tolerate lung resection. However, these tests are rarely carried out.

Q. Why should patients for lobectomy be evaluated for suitability for pneumonectomy?

Ans. Patients for lobectomy should be evaluated for suitability for pneumonectomy for the following reasons:

- During the immediate postoperative period, contamination with blood or secretions, atelectasis, and infection can worsen the function of the lung tissue remaining on the operative side, thereby presenting like a functional pneumonectomy.
- Intraoperative reassessment of the disease may change the staging and may result in change of the surgical plan from lobectomy to pneumonectomy.

Hence, patients scheduled for lobectomy must be assessed for the ability to tolerate pneumonectomy as well. Patients not meeting the criteria for pneumonectomy require careful surgical planning and meticulous attention to optimizing the residual lung function.

Q. Summarize the preoperative cardiorespiratory assessment of a patient presenting for pneumonectomy.

Ans. No single test of respiratory function can be used as a method of preoperative assessment. An estimate of respiratory function in all three areas (testing of lung mechanics, parenchymal function, and cardiopulmonary

reserve)—the so-called three-legged approach—should be made for each patient. Transport of oxygen to the lungs correlates with respiratory mechanics, transport into the blood depends on the parenchymal function, and transport to the tissues relies on cardiopulmonary interaction.

All patients undergoing lung resection should have spirometry and DLCO testing done to calculate predicted postoperative lung function. Assessment of effort tolerance should be done to evaluate cardiac function. Patients with borderline ppo lung functions should be considered for V/Q scanning. Patients with a history of cardiorespiratory disease, patients with limited effort tolerance, and patients with borderline ppo lung functions should undergo ABG analysis and cardiopulmonary exercise testing. A stepwise algorithm for evaluation for lung resection is outlined in **Flowchart 1**.

Q. Why is preoperative respiratory optimization important in this patient?

Ans. Patients presenting for lung resection are at high risk for perioperative respiratory complications such as atelectasis and pneumonia because of three reasons:

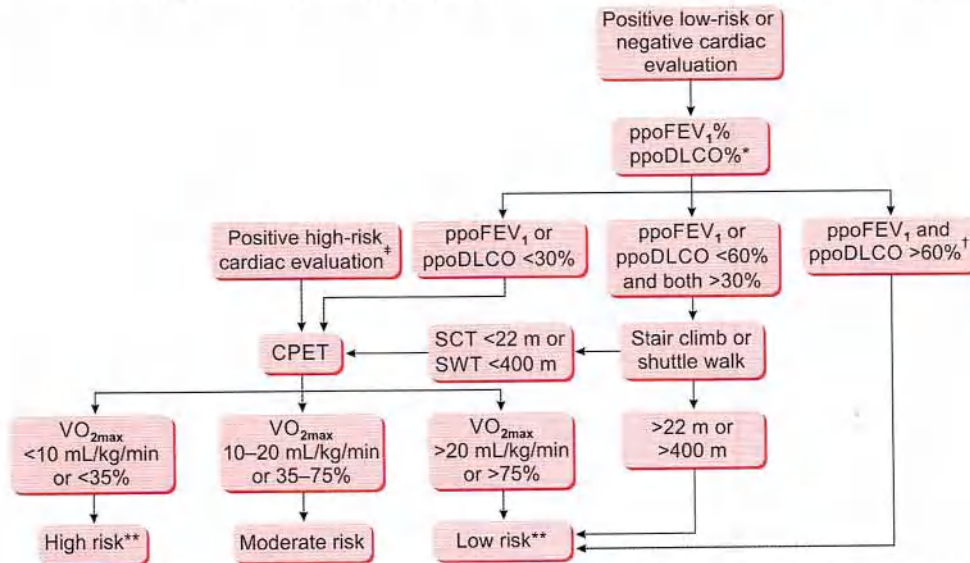
1. Most of these patients are smokers and have underlying pre-existing respiratory disease.
2. Handling of the lung during surgery may cause dysfunction of the surrounding normal tissue.
3. If a thoracotomy approach is used, postoperative pain may interfere with the patient's ability to cough and perform spirometry.

Therefore, it is important to maximally optimize preoperative respiratory function to minimize complications.

Q. What is the preoperative optimization of this patient?

Ans. The five elements of the preoperative regimen are stopping of smoking, dilating airways, loosening and removing secretions, taking measures to increase motivation and education, and facilitating postoperative care.

Cessation of smoking is the single most important intervention in the preoperative period. A study by Warner found that the incidence of postoperative pulmonary complications was highest (57%) in patients who quit smoking for <8 weeks; it decreased to 14.5% if cessation was >8 weeks. It was least (11.9%) in those who never smoked. Current smokers (33%) also had a high risk of postoperative complications. It is hypothesized that the absence of the irritant effect of cigarette smoke in the postoperative period inhibits coughing and leads to retention of secretions and small airway obstruction, in patients who stopped smoking for <8 weeks. Within 12 hours of cessation, the carboxyhemoglobin levels drop. Long-term cessation (>8 weeks) leads to improvement in mucociliary function with increased sputum clearance and also reduced airway

Flowchart 1: Algorithm for pulmonary preoperative assessment of patients requiring lung resection.

(CPET: cardiopulmonary exercise testing; ppoDLCO: predicted postoperative diffusing capacity for carbon monoxide; ppoDLCO%: percent predicted postoperative diffusing capacity for carbon monoxide; ppoFEV₁: predicted postoperative FEV₁; ppoFEV₁%: percent predicted postoperative FEV₁; SCT: stair climb test; SWT: shuttle walk test; VO_{2max}: maximal oxygen consumption).

*For pneumonectomy candidates, it is suggested to use perfusion scan to calculate predicted postoperative values of FEV₁ or DLCO [PPO values = preoperative values × (1 – fraction of total perfusion for the resected lung)], where the preoperative values are taken as the best measured postbronchodilator values. For lobectomy patients, segmental counting is indicated to calculate predicted postoperative values of FEV₁ or DLCO (PPO values = preoperative values × (1 – y/z), where the preoperative values are taken as the best measured postbronchodilator value and the number of functional or unobstructed lung segments to be removed is y and the total number of functional segments is z).

†ppoFEV₁ or ppoDLCO cutoff values of 60% predicted values have been chosen based on indirect evidences and expert consensus opinion. Cardiac risk is determined using the thoracic revised cardiac risk index.

*For patients with a positive high-risk cardiac evaluation deemed to be stable to proceed to surgery, it has been suggested to perform both PFTs and cardiopulmonary exercise test for a more precise definition of risk.

**Definition of risk: *Low risk*—the expected risk of mortality is <1%. Major anatomic resections can be safely performed in this group. *Moderate risk*—morbidity and mortality rates may vary according to the values of split lung functions, exercise tolerance, and extent of resection. The risks and benefits of the operation should be thoroughly discussed with the patient. *High risk*—the risk of mortality after standard major anatomic resections may be >10%. Considerable risk of severe cardiopulmonary morbidity and residual functional loss is expected. Patients should be counseled about alternative surgical (minor resections or minimally invasive surgery) or nonsurgical options.

Source: Brunelli A, Kim AW, Berger KI, Addrizzo-Harris DJ. Physiologic of the patient with lung cancer being considered for resectional surgery: Diagnosis and management of lung cancer, 3rd edition: American College of Chest Physicians evidence-based clinical practice guidelines. Chest. 2013;143:e1665.

reactivity and sputum production. The risk for patients who quit for >6 months is the same as those who never smoked. Smoking has also been shown to delay wound healing.

Bronchodilator therapy will benefit not only these patients but also those without bronchospasm even if no improvement in FEV₁ is seen.

Steroids: Inhaled corticosteroids improve symptoms and lung function and reduce exacerbations when FEV₁ is <60% of the predicted value.

Many of these patients might be receiving steroids. The recommendations for perioperative management of patients on steroid are given in **Table 2**.

Patients with cor pulmonale may be receiving *diuretics and/or digitalis*. This will need to be continued perioperatively.

Hydration and mucolytic therapy will help in loosening of secretions and along with chest physiotherapy they will help in clearing the chest.

Preoperative *incentive spirometry* has always been recommended for reducing postoperative pulmonary complications though there is no supportive evidence.

Q. What anesthetic technique would you use?

Ans. General anesthesia with controlled ventilation with thoracic epidural analgesia should be used. Intravenous induction with propofol or thiopentone can be performed. Propofol is preferred since many of these patients will have reactive airways and use of thiopentone and tracheal instrumentation in light plane can lead to bronchospasm. Since this patient has a normal airway, a nondepolarizing neuromuscular blocking agent can be used to facilitate

TABLE 2: Recommendations for perioperative management of patients on steroids.

Dose	Surgery	Recommended dose
<10 mg prednisolone or equivalent/day	Minor/moderate/major	Additional steroid cover not required (assume normal HPA response)
>10 mg prednisolone or equivalent/day	Minor surgery	25 mg of hydrocortisone at induction and normal medications postoperatively
>10 mg prednisolone or equivalent/day	Moderate surgery	Usual dose preoperative and 25 mg hydrocortisone IV at induction, then 25 mg IV TDS for 1 day, then recommence preoperative dosage
>10 mg prednisolone or equivalent/day	Major surgery	Usual dose preoperative and 100 mg hydrocortisone at induction, then 100 mg IV TDS for 2–3 days

(HPA: hypothalamic-pituitary-adrenal).

intubation and intermittent positive pressure ventilation (IPPV).

Maintenance of anesthesia: The two factors influencing the decision of the choice of anesthetic agents for maintenance of anesthesia are hypoxemia during one-lung ventilation (OLV) and risk of acute lung injury secondary to release of proinflammatory mediators after re-expansion of the collapsed lung. The 2013 Cochrane review intravenous versus inhalation anesthesia for OLV found no evidence to indicate that the drug used to maintain anesthesia during OLV affected participant outcomes. Though most studies conclude that there is no significant difference in oxygenation between intravenous and inhalational techniques (with or without epidural anesthesia), there is some evidence regarding superiority of sevoflurane anesthesia over total intravenous anesthesia (TIVA) regarding proinflammatory mediators.

Q. What monitors would you like to use for this patient?

Ans. Monitoring will include ECG, pulse oximetry, capnography, noninvasive blood pressure, and temperature probe. Urinary catheterization is not indicated in straightforward lobectomies but maybe needed in complicated lobectomies and in pneumonectomies which are associated with more hemodynamic instability. Invasive arterial pressure monitoring is useful in patients who are at higher cardiopulmonary risk and in those in whom the surgery is expected to be complicated. An arterial cannula also allows blood gas sampling during OLV. Central venous pressure monitoring may be indicated in patients with serious comorbidities for intravascular volume assessment as well as administration of vasoactive drugs.

Q. When is a pulmonary artery (PA) catheter indicated? What are the problems with PA catheter in left lateral decubitus position?

Ans. Pulmonary artery catheters are not routinely indicated in pneumonectomy. They are potentially useful to monitor pulmonary artery pressure, right ventricular function,

and left heart filling pressures in the postoperative period. The data is less reliable intraoperatively with an open hemithorax. They are difficult to position and may require fluoroscopic guidance. They are also associated with significant risks such as injury to the fresh suture line on the pulmonary artery on the operated side and incorrect readings due to malposition and intrinsic limitations such as obstruction to the blood flow into the left atrium by an inflated balloon.

Pulmonary artery catheters usually tend to locate in the right lung. Hemodynamic measurements are most accurate when the tip of the PA catheter is situated in West zone 3. During a right thoracotomy with OLV, the catheter tip is usually in the nondependent lung. This lung is collapsed and not ventilated, and therefore the hemodynamic measurements may be inaccurate.

Q. What is the plan for ventilation intraoperatively?

Ans. The patient will need lung separation and selective ventilation of the left lung to allow collapse of the right lung and access to the bronchovascular structures.

Q. What are the indications for lung separation?

Ans. The indications for lung separation can be classified as follows:

- Separation of one lung from the other to prevent spillage or contamination:
 - Unilateral bronchopulmonary lavage in patients with pulmonary alveolar proteinosis
 - Lung abscess
 - Bronchiectasis
 - Pulmonary hemorrhage
- Control of distribution of ventilation (differential lung ventilation):
 - Bronchopleural fistula
 - Surgical opening of major bronchus
 - Giant unilateral cyst or bulla
- Surgical exposure (unilateral lung collapse and OLV):
 - Lung, esophageal, vascular, mediastinal, spine surgeries in the thoracic cavity.

TABLE 3: Advantages and disadvantages of various techniques for lung isolation.

Techniques	Advantages	Disadvantages
Double-lumen tube	• Can suction lungs independently • Quality of suctioning better • Can apply CPAP to nonventilated lung more easily • Easy to switch ventilation from one lung to the other • Preferred when sleeve resection of bronchus planned	• Difficult to insert in distorted airway and in patients at risk of aspiration • Needs change of tube if postoperative ventilation is considered, which may be risky • Needs determination of appropriate size • Potential for tracheobronchial injury
Bronchial blocker	• Can be used for selective lobar blockade • Can be used in tracheostomized patients • Can be used in critically ill patients who are already intubated with a single-lumen tube • Can be used in children and small adults, in whom the smallest DLT maybe too big	• Because of small lumen, lung inflates and deflates very slowly • More difficult to apply CPAP to nondependent lung • Cannot be used if the main stem bronchus on the operative side is involved by disease
Endobronchial tube	• Useful in emergencies such as massive bleeding • Useful in children and very small adults	• Inability to ventilate or suction the opposite lung • If on the right side, high risk of obstructing the right upper lobe bronchus • Difficult to negotiate into the left side

(CPAP: continuous positive airway pressure; DLT: double-lumen endotracheal tube).

Q. Describe various techniques for lung isolation with advantages and disadvantages of each.

Ans. Three types of devices are available for providing OLV during anesthesia—double-lumen endotracheal tubes (DLTs), bronchial blockers, and endobronchial tubes (Table 3).

Q. How is the size of the DLT determined for each patient?

Ans. Single-use polyvinyl chloride DLTs are available in sizes 26, 28, 32, 35, 37, 39, and 41 Fr.

A well-sized DLT should pass easily through the glottis and should enter the intended main bronchus without causing trauma. Excessive pressures should not be needed for inflating the bronchial cuff; at the same time, an air leak should be present when the bronchial cuff is deflated.

Generally, 35 and 37 Fr are used for small and large females, respectively, and 39 and 41 Fr for small and large males, respectively.

Measurement of the tracheal diameter on the preoperative CT scan has been used to predict the correct size of the DLT needed for a patient (Table 4).

TABLE 4: Ratio of double-lumen endotracheal tube (DLT) size with measurement of tracheal width.

Measured tracheal width (mm)	DLT size (Fr)
≥18	41
≥16	39
≥15	37
≥14	35

Source: Brodsky JB, Macario A, Mark JB. Tracheal diameter predicts double-lumen tube size: a method for selecting left double-lumen tubes. *Anesth Analg*. 1996;82:861-4.

It has also been found that tracheobronchial dimensions correlate with height. Therefore, the height of the patient can be used to choose the size of the DLT (Table 5). The depth of insertion can be calculated as $12 + (\text{patient height}/10)$ cm. It should be noted that most of the formulae quoted in western literature, both for the size of the DLT and for the depth of insertion, are derived from Caucasian populations and may not apply to Asian patients with smaller build.

For females <152 cm, consider 32 Fr. For males <160 cm, consider 37 Fr.

TABLE 5: Ratio of double-lumen endotracheal tube (DLT) size with height of the patient.

Sex	Height (cm)	DLT size (Fr)
Male	>170	41
Male	<170	39
Female	>160	37
Female	<160	35

Q. Why are left-sided DLTs preferable to right-sided ones?

Ans. The right upper lobe bronchus takes off from the right main bronchus 1.5–2 cm below the carina. Therefore, when a right-sided DLT is placed, there are high chances that the right upper lobe bronchus maybe occluded, and care has to be taken to align the slot for the right upper lobe bronchus, with the opening of the bronchus.

The left mainstem bronchus is much longer than the right one (50 mm as compared to 25 mm). Therefore, there is a greater margin of safety while positioning a left-sided DLT.

Q. Are there any specific indications for right-sided DLTs?

Ans. Specific conditions where a right-sided DLT would be indicated are as follows:

- Exophytic tumor that compresses the entrance of the left bronchus
- Intraluminal tumor near the entrance of the left bronchus
- Left-sided tracheobronchial disruption
- Left bronchus stent
- Descending thoracic aortic aneurysm compressing the entrance of the left bronchus
- Sharp angle of the entrance of the left bronchus.

Q. You decide to insert a 39 Fr right DLT in this patient. Describe the technique of insertion and confirmation.

Ans. The DLT is passed with the distal curvature initially concave anteriorly. After the tube tip passes the larynx, the stylet is removed and the tube is rotated 90° to the right (so that the distal curve is now concave toward the right side and the proximal curve is concave anteriorly) to allow endobronchial intubation on the right side. After rotation, the tube is advanced until most of it is inserted and resistance to further insertion is felt. The average depth of insertion for both male and female patients 170 cm tall is 29 cm, and for each 10 cm increase or decrease in height, the average placement depth is increased or decreased by 1 cm.

After insertion, the tracheal cuff should be inflated, and both lungs should be auscultated for equal ventilation. If breath sounds are not equal, the tube is probably too far in, and the tracheal lumen opening is in a main stem bronchus. The tube should then be withdrawn until breath sounds are bilaterally equal.

The second step is to clamp the tracheal lumen (left side in the case of the right-sided tube) and open the tracheal cap. Then, the bronchial cuff is slowly inflated such that there is no air leak from the bronchial lumen around the bronchial cuff into the tracheal lumen. This ensures that the bronchial cuff is not excessively inflated. Inflation of the bronchial cuff usually requires 2–3 mL of air.

The third step is to remove the clamp and check that both lungs are ventilated with both cuffs inflated. This ensures that the bronchial cuff is not obstructing the contralateral bronchus, either totally or partially.

The final step is to clamp each side selectively and watch for absence of movement and breath sounds on the ipsilateral (clamped) side; the ventilated side should have clear breath sounds, chest movement that feels compliant and peak airway pressures should not be excessively high.

The pediatric flexible fiberoptic bronchoscope is the gold standard for confirmation of DLT position. When available, the bronchial cuff should be inflated under

vision to prevent the risk of inflating it within a secondary bronchus in a malpositioned DLT (risk of rupture of the bronchus). When using a right-sided DLT, the bronchoscope is usually first introduced through the tracheal lumen. The carina is visualized, but no bronchial cuff herniation should be seen. The upper surface of the blue endobronchial cuff should be just below the tracheal carina. The bronchoscope should then be passed through the bronchial lumen, and the right middle and lower lobe bronchial orifices should be identified. The orifice of the right upper lobe bronchus should be identified when the bronchoscope is passed through the right upper lobe ventilating slot of the DLT.

Q. Can a bronchial blocker be used in this patient? What precautions are needed during surgery?

Ans. This patient will need a left-sided bronchial blocker. Since the left main bronchus is affected by disease, it will not be possible to place the blocker. There are high chances of malposition, trauma to the bronchus, and dislodgement and distal embolization of tumor.

When a bronchial blocker is used for a pneumonectomy, care should be taken to withdraw the blocker prior to stapling the bronchus.

Q. The surgery will be done in lateral decubitus position. Describe how the lateral decubitus position is given and what are the problems with this position.

Ans. Following confirmation of the side of surgery, the patient is turned into the lateral position. The lower shoulder is pulled through anteriorly. The lower arm is flexed at the elbow and tucked under the pillow supporting the head. The upper arm is extended and placed over the head on an armrest.

The lower leg is slightly flexed at the hip and knee while the upper leg is kept relatively straight. A pillow is placed between the legs. Chest and pelvic supports are used for further stability. All vulnerable areas are padded. A small roll is placed under the dependent thorax to prevent pressure on the dependent arm neurovascular bundle. This padding should not be allowed to migrate into the axilla where it can worsen the pressure on the neurovascular bundle. The cervical spine should be aligned with the thoracolumbar spine, and there should be no pressure on the lower eye.

The common problems with this position are as follows:

- Ischemia, nerve damage, or compartment syndrome to the dependent arm
- Postoperative shoulder discomfort
- Lateral angulation of the neck leading to jugular venous obstruction
- Hyperextension of the nondependent arm leading to traction or compression of the brachial and axillary neurovascular bundles.

(Please refer to any standard anesthesia textbook for understanding the physiology of OLV).

Q. Describe the management of one-lung ventilation.

Ans. Before instituting one-lung ventilation:

Nondependent lung: The composition of the gas mixture in the nondependent lung will affect the rate at which the lung collapses. The presence of nitrogen, which has low blood gas solubility, will delay collapse. This can be avoided by ventilating the lung with 100% oxygen prior to instituting OLV. Collapse of the nondependent lung can also be improved by inflating the bronchial cuff after a short period of apnea.

Dependent lung: To prevent atelectasis of the dependent lung, a recruitment maneuver should be applied immediately before starting OLV.

During one-lung ventilation:

- Maintain tidal volume 5–6 mL/kg:
 - Maintain peak airway pressure <35 cm H₂O
 - Maintain plateau pressure <25 cm H₂O.
- Positive end-expiratory pressure of 5 cm H₂O to ventilated lung:
 - Avoid in patients with COPD
- Respiratory rate to maintain normal PaCO₂
- Volume- or pressure-controlled mode.

Q. After switching to OLV, the patient's saturation drops to 90%. What is the management of hypoxemia during OLV?

Ans. If hypoxemia occurs during OLV, the following protocol should be followed:

- *For sudden or severe desaturation:* Convert to two-lung ventilation.
- *For gradual desaturation:*
 - Increase FiO₂ to 1.0.
 - The position of the DLT should be rechecked using a fiberoptic bronchoscope. If a left thoracotomy is being performed using a right-sided DLT, ventilation to the right upper lobe should be ensured.
 - The hemodynamic status of the patient should be optimized.
 - Recruitment of the ventilated lung.
 - Positive end-expiratory pressure (PEEP) of 5–10 cm H₂O can be applied to the dependent lung.
 - Continuous positive airway pressure (CPAP) of 1–2 cm H₂O should be applied to the nondependent lung, after a recruitment maneuver.
 - Intermittent two-lung ventilation can be reinstituted after discussion with the surgeon.
 - Partial ventilation of the nonventilated lung using either low-flow oxygen in insufflations or high-frequency ventilation.
 - If a pneumonectomy is being performed, ligation of the pulmonary artery can be carried out to comple-

tely eliminate the shunt. Looping the ipsilateral pulmonary artery (if technically feasible) will also reduce shunt in a nonpneumonectomy surgery.

Q. What is the plan for fluid management and why is this so important in this patient?

Ans. Fluid restriction is generally advocated in lung resections. Maintenance fluid should be given at a rate of 2 mL/kg/h. This is particularly important in right pneumonectomies where a significant portion of the pulmonary vascular bed is resected. The reasons for this are as follows:

- Third spacing is not excessive in lung surgeries.
- The dependent lung has a tendency for high capillary hydrostatic pressures.
- Postoperative pulmonary edema may occur if the remaining pulmonary vasculature cannot accommodate the entire cardiac output. This is true, especially in patients with long-standing respiratory disease whose pulmonary vasculature is less distensible. This complication is associated with a high mortality.
- Intraoperative lung manipulation and collapse may impair lymphatic drainage.
- Pneumonectomy increases the afterload to the right heart due to rise in pulmonary artery pressure. An additional volume overload may increase the risk of right heart failure.

It is recommended that the total positive fluid balance in the first 24 hours should not exceed 20 mL/kg².

Q. Will this patient need ventilation postoperatively?

Ans. If the patient is awake, warm, pain-free with acceptable hemodynamic and respiratory parameters, it is advisable to extubate the patient in the operation theater. Positive pressure ventilation is best avoided after lung resection to prevent tension on the suture lines and chances of a stump blow-out. Positive pressure ventilation also increases the pulmonary vascular pressure. If ventilatory support is needed, use of pressure support ventilation is more appropriate. Patients with borderline lung functions may benefit from a gradual weaning process.

Q. Why is the insertion of chest drain in the postpneumonectomy space (PPS) controversial?

Ans. It is crucial that the mediastinum remains in the center in the postoperative period as a significant mediastinal shift can lead to cardiovascular collapse. While controlled drainage of air and fluid may be needed to avoid shift to the opposite side, excessive drainage can have the opposite effect. There is no consensus among thoracic surgeons regarding insertion of chest drain in these patients. The common practice in pneumonectomy patients is either to insert the chest drain and keep it clamped or to not insert the chest drain at all,

the latter favored in uncomplicated cases. The problems associated with chest drain are risk of infection and risk of inadvertent application of negative pressure to the drain. (Low pressure suction is a standard practice in lung resection surgery in many centers for drainage of air and improved lung expansion. This can be disastrous in the PPS as it can cause a large mediastinal shift and cardiovascular collapse.) The chest drain, if inserted, is unclamped intermittently to look for bleeding or rarely air leak.

Q. What are the available techniques for pain relief in this patient?

Ans.

Thoracic epidural analgesia is considered the gold standard for post-thoracotomy analgesia. The epidural is most effective when placed at the vertebral level corresponding with the dermatomes of the surgical incision. Local anesthetic solutions may be infused continuously or via a patient-controlled device. The side effects of the local anesthetic may be reduced by adding opioids to the solution. Other options can be considered when thoracic epidural analgesia is not feasible or is contraindicated. These include:

- *Parenteral opioids:* They can provide adequate analgesia. The side effects include respiratory depression, sedation, and loss of cough. Patient-controlled analgesia (PCA) devices are used to deliver opioids in a more patient-specific way and are superior to intravenous boluses or intramuscular injections.
- *Paravertebral blocks:* A catheter may be placed in the paravertebral space under direct vision by the surgeon or percutaneously by the anesthetist, and plain local anesthetic solution infused continuously, blocking intercostal nerves above and below as they pass extrapleurally through the space. The main advantage over thoracic epidurals is that the block is unilateral, thus reducing the extent of related side effects. However, large volumes of local anesthetic solution are required, and the risk of local anesthetic toxicity is increased.
- *Intrathecal opioids:* Preservative-free morphine may be injected via a spinal needle into the subarachnoid space at the lumbar spine level. As it is less lipid soluble than fentanyl or diamorphine there is rostral spread in the cerebrospinal fluid (CSF), and a single-shot dose of 5–20 µg/kg may provide analgesia for thoracic surgery for up to 24 hours postoperatively. Delayed respiratory depression has been reported.
- *Intercostal nerve blocks:* Local anesthetic solutions may be injected into the intercostal spaces above and below the surgical incision. The main disadvantage is the short-acting nature of this technique. It may be used to supplement other techniques or provide rapid-onset relief until another technique has been established.

A summary of the various techniques available for post-thoracotomy pain relief is available from the PROSPECT (procedure specific postoperative pain management) guidelines.

Q. What postoperative complications can occur after pneumonectomy?

Ans. Complications are more common after a right pneumonectomy than a left one due to the greater physiological disturbance.

- *Cardiovascular:*
 - Arrhythmias—atrial fibrillation is most common. Amiodarone should be used with caution due to its pulmonary toxicity.
 - Right ventricular failure.
 - Cardiovascular collapse due to cardiac herniation (more likely when part of pericardium is excised) or torsion of major vessels due to gross mediastinal shift.
 - Hemorrhage—more common in pleuropneumectomy.
- *Pulmonary:*
 - Pulmonary edema
 - Respiratory insufficiency
 - Pulmonary torsion
 - Postpneumonectomy syndrome—usually a late complication caused by compression of the trachea or bronchus due to mediastinal shift and the hyperinflation of lung.
- *Pneumonectomy space:*
 - Bronchopleural fistula
 - Empyema
 - Disruption of the bronchial stump causing tension pneumothorax.
- *Neurological:* Recurrent laryngeal, vagus, or phrenic nerve injury.

Q. What are the changes in the PPS?

Ans. Immediately after a pneumonectomy, the hemithorax is filled with air. Within 24 hours, the ipsilateral hemidiaphragm becomes slightly elevated, the mediastinum shifts slightly toward the PPS, and fluid starts accumulating in the PPS. Fluid generally accumulates at a rate of 1–2 intercostal spaces per day in the immediate postoperative period. As pleural pressure increases, the rate of fluid accumulation decreases, and after 2 weeks, 80–90% of the PPS is filled with fluid. On chest radiograph, complete opacification of the hemithorax after pneumonectomy takes an average of 4 months, with a range from 3 weeks to 7 months. The differential diagnosis of rapid accumulation of fluid into the PPS in the immediate postoperative period includes hemorrhage, infection, or development of a chylothorax.*

*UpToDate. Sequelae- and-complications-of-pneumonectomy. [online] Available from <https://www.uptodate.com/contents/sequelae- and-complications-of-pneumonectomy#H2>. [Last accessed February, 2020].

Q. One week after surgery, this patient presents with dyspnea, subcutaneous emphysema, contralateral deviation of the trachea, and persistent air leak from the chest tube. Bronchoscopy shows a bronchopleural fistula. This patient is posted for surgical closure of the fistula with flap cover. What are the goals of anesthetic management?

Ans. The goals of anesthetic management include maintenance of adequate ventilation and oxygenation and protection of the contralateral lung from spillage of purulent material.

Anesthetic strategies for induction of anesthesia depend on the severity of the bronchopleural fistula and the presence of infection:

- A chest drain is necessary to prevent accumulation of escaping air leading to a tension pneumothorax and to drain purulent contents to minimize contamination.
- Positive pressure ventilation may be ineffective because of loss of tidal volume through the fistula.
- In addition, positive pressure ventilation carries the risk of contaminating the noninfected lung.

The options for induction of anesthesia include:

- Spontaneous ventilation and induction using a volatile anesthetic (sevoflurane), and intubation with a double-lumen tube. The patient should be positioned head-up with the contaminated lung down to minimize contamination of the healthy lung.
- Rapid-sequence induction to minimize the time before tracheal intubation and lung isolation.
- Awake intubation in patients at increased risk for aspiration of gastric contents caused by full stomach or bronchopleuroenteric fistula.

Once the tube has been positioned, the healthy lung should be isolated immediately and the head elevated slightly to decrease the likelihood of contamination. In patients with a small chronic bronchopleural fistula with minimal air leak and no associated infection or empyema, a standard endotracheal tube can be used safely. Anesthesia is maintained by IPPV delivered to the healthy lung. (In nonpneumonectomy cases, if the affected lung needs to be ventilated, low tidal volume ventilation or CPAP or high-frequency jet ventilation may be tried.) Emergence should be smooth with avoidance of high airway pressures. Pressure support ventilation may be preferred if the patient needs postoperative ventilatory support.

Q. Describe your anesthesia plan if this patient with bronchopleural fistula requires anesthesia for insertion of an intercostal drain.

Ans. Usually, these patients will be pale (hypotensive, hypoxic), uncooperative, and/or combative. It is important to maintain spontaneous ventilation at all times as positive pressure ventilation is associated with volume loss during tidal ventilation. If possible, the patient should be positioned with back-up and procedure carried out under local anesthesia. If this is not possible, the patient may be given small doses of ketamine (1–1.5 mg/kg) while preserving spontaneous breathing. This will ensure hemodynamic stability and preservation of reflexes.

SUGGESTED READING

1. Alexander D, Keogh B. Preoperative assessment for thoracic surgery. *Anaesth Intensive Care Med.* 2005;6:386–8.
2. American Thoracic Society. ATS statement: guidelines for the six-minute walk test. *Am J Respir Crit Care Med.* 2002;166:111–7.
3. British Thoracic Society. Society of Cardiothoracic Surgeons of Great Britain and Ireland Working Party. BTS guidelines: guidelines on the selection of patients with lung cancer for surgery. *Thorax.* 2001;56:89–108.
4. Brodsky JB, Macario A, Mark JB. Tracheal diameter predicts double-lumen tube size: a method for selecting left double-lumen tubes. *Anesth Analg.* 1996;82:861–4.
5. Brunelli A, Charloux A, Bolliger CT, Rocco G, Sculier JP, Varela G, et al. European Respiratory Society and European Society of Thoracic Surgeons joint task force on fitness for radical therapy. ERS/ESTS clinical guidelines on fitness for radical therapy in lung cancer patients (surgery and chemoradiotherapy). *Eur Respir J.* 2009;34:17–41.
6. Campos JH. Which device should be considered the best for lung isolation: double-lumen endotracheal tube versus bronchial blockers. *Curr Opin Anaesthesiol.* 2007;20:27–31.
7. Pierce RJ, Copland JM, Sharpe K, Barter CE. Preoperative risk evaluation for lung cancer resection: predicted postoperative product as a predictor of surgical mortality. *Am J Respir Crit Care Med.* 1994;150:947–55.
8. Slinger PD, Campos JH. Anesthesia for thoracic surgery. In: Miller RD (Ed). *Miller's Anaesthesia*, 9th edition. Philadelphia: Churchill Livingstone; 2020.
9. The European Society of Regional Anaesthesia and Pain Therapy. Procedure specific postoperative pain management. Better postoperative pain management. [online] Available from <https://esraeurope.org/prospect/>. [Last accessed February, 2020].
10. Walker C. Analgesia after thoracotomy. *Anaesth Intensive Care Med.* 2005;6:418–20.

Bronchiectasis with Lung Abscess

Priya Ranganathan, Amol Trimbakrao Kothekar, Swapnil Parab

A 32-year-old woman presented with the history of fever, cough with purulent sputum and progressively worsening dyspnea since 2 months. She has history of pulmonary tuberculosis 6 years ago, for which antitubercular therapy was taken. Subsequently, she was diagnosed to have left lower lobe bronchiectasis. She was treated for left lower lobe pneumonia 2 months ago. She has no other medical illnesses. On examination, her height was 150 cm and weight was 56 kg. She has clubbing. There are bronchial breath sounds on the left side with basal coarse crepitations. This patient is posted for left lower lobectomy.

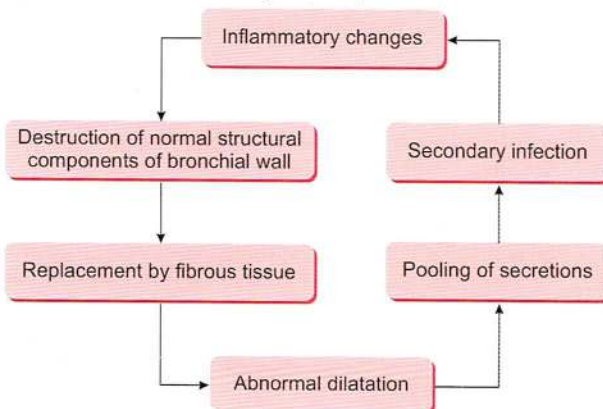
Q. What is bronchiectasis?

Ans. Bronchiectasis is defined as abnormal and irreversible dilatation of part of the bronchial tree, usually at the level of the segmental or subsegmental bronchi.

Q. What are the pathological changes in bronchiectasis?

Ans. The pathological changes in bronchiectasis are given in **Flowchart 1**.

Flowchart 1: Pathological changes in bronchiectasis.



Q. What is the etiology of bronchiectasis?

Ans. Etiologically bronchiectasis can be categorized as cystic fibrosis bronchiectasis and noncystic fibrosis bronchiectasis.

Bronchiectasis could be due to:

1. Infectious causes—Pneumonia due to:
 - a. Bacterial infections, e.g., *Haemophilus influenzae*, *Mycobacterium tuberculosis*, *Mycobacterium avium* complex, *Staphylococcus aureus*, and anaerobes
 - b. Viral infections, e.g., adenovirus and influenza virus.
2. Noninfectious causes:
 - a. Aspiration pneumonia
 - b. Airway obstruction
 - c. Immune reactions—especially after allergic bronchopulmonary aspergillosis.

Q. What are the predisposing factors for bronchiectasis?

Ans.

Predisposing factors include:

- Immunosuppression—HIV infection, immunoglobulin deficiency
- Disorders of ciliary motility—primary ciliary dyskinesia, (including Kartagener's syndrome)
- Disorders of mucus secretion—cystic fibrosis.

Q. What are the clinical features of bronchiectasis?

Ans.

Symptoms:

- Persistent or recurrent productive cough
- Purulent sputum
- Frequent respiratory tract infections
- Hemoptysis
- Nonspecific symptoms like fatigue, weight loss.

Signs:

- Variable auscultatory findings like crepitations, wheeze, and bronchial breath sounds
- Clubbing
- Long-standing cases may have signs of cor pulmonale and right ventricular failure.

Q. What investigations would you like to order for this patient?**Ans.**

- **Complete blood count (CBC):** There may be raised white blood cell (WBC) count. Neutrophil predominance if bacterial infection, lymphocytic predominance if tuberculous. Anemia may be present in view of chronic illness.
- **Liver function test (LFT) and renal function test (RFT):** In view of major surgery and chronic illness.
- Fibrinogen level correlates with the severity of bronchiectasis and higher fibrinogen (no cut-off value) is associated with worse lung function.
- **Chest X-ray (CXR):** It is done to check extent of bronchiectasis, rule out cavitory lesion, rule out relapsed tuberculosis (TB), rule out pleural effusion, check position of trachea and bronchi in view of possible fibrosis with shift of trachea—since patient is presenting for lobectomy and will need lung isolation. Not needed if CT thorax done.
- **Computed tomography (CT) thorax:** To check the extent of bronchiectasis and lung abscess. When CT chest is done, CXR is not required. Measuring diameter of trachea and bronchi may be useful to decide size of double-lumen tube (DLT).
- **Electrocardiogram (ECG):** To check for signs of pulmonary hypertension, which include presence of tall peaked P wave (P pulmonale), tall R wave in V1, right bundle branch block (RBBB), right axis deviation. Right ventricular strain pattern [ST depression with T wave inversion in the right-sided precordial leads (RV1–RV3)] and also in the inferior leads more prominent in lead III.
- **Pulmonary function test [spirometry and diffusing capacity of lung for carbon monoxide (DLCO)]:** To document severity of dysfunction, to check for response to bronchodilator therapy and to assess suitability for lung resection.
- **Room air oxygen saturation (SpO₂):** To check for baseline hypoxemia.
- **Arterial blood gas (ABG):** For hypoxia, high partial pressure of carbon dioxide (PCO₂) along with raised bicarbonate (HCO₃) and normal pH suggest coexisting chronic obstructive pulmonary disease (COPD).

- **Two-dimensional echocardiography:** To measure pulmonary artery pressure to diagnose pulmonary hypertension and right ventricular size (hypertrophy in long-standing pulmonary hypertension).
- **Sputum, Gram's stain, culture and sensitivity:** To identify infectious organisms and to guide appropriate antibiotic therapy. Many patients are colonized with *Pseudomonas aeruginosa*.

Q. What are the high-resolution chest computed tomography (HRCT) scan specific criteria for bronchiectasis?**Ans.** Specific criteria include:

- The internal diameter of the bronchus is larger than diameter of its accompanying vessel.
- The bronchus fails to taper in the periphery of the chest. Airway wall thickening is not diagnostic of bronchiectasis.
- Airway visibility within 1 cm of costal pleural surface or touching mediastinal pleura.

Q. All blood investigations and a 12-lead ECG were within normal limits. CT of the chest revealed fibrocavitary and bronchiectatic changes in the left lower lobe with suspicion of a left lower lobe abscess. The right lung was relatively normal. Spirometry showed the following values. Interpret these values. Will this patient benefit from preoperative bronchodilator therapy?

Ans. Spirometry shows a reduction in forced expiratory volume in 1 second (FEV1), though forced vital capacity (FVC) is normal. FEV1/FVC is reduced. This suggests moderate obstructive pattern. Postbronchodilator, there is an improvement in FEV1 by 330 mL, which is about 12% of the baseline value. Changes in FEV1 by >12% or 200 mL are taken as indicators of good reversibility. Therefore, this patient will benefit from preoperative bronchodilator therapy. Since spirometry values depend on patient performance, they may not be representative of true underlying state when patient's efforts are suboptimal. Therefore, patients who do not appear to have good reversibility on spirometry may also benefit from perioperative bronchodilator therapy, if it is clinically indicated (Table 1).

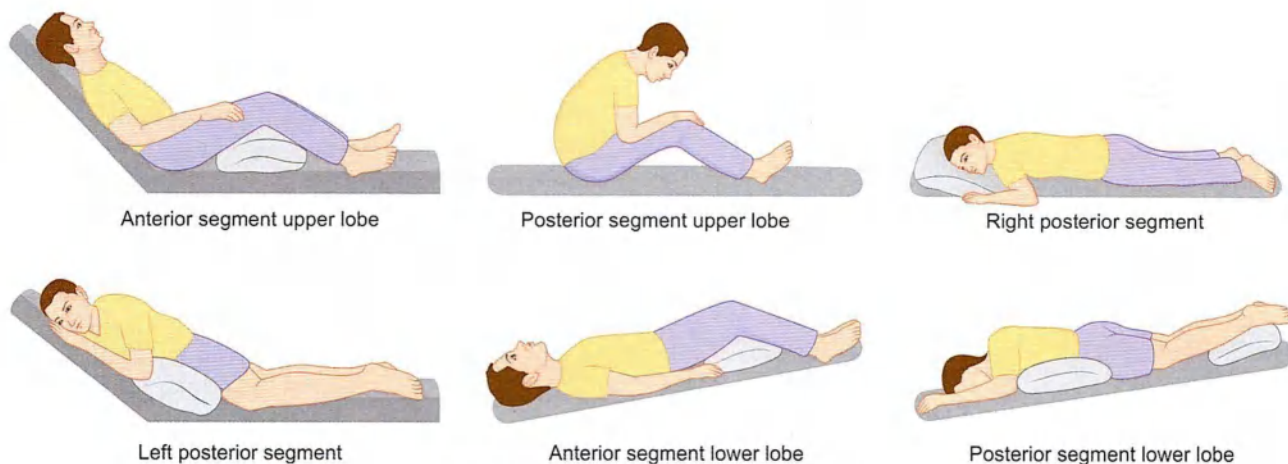
Q. What are the preoperative preparations of a patient with bronchiectasis presenting for surgery?**Ans.** The preoperative preparation consists of:

- Cessation of smoking (if patient is smoker). Refer to Chapter 10 on COPD for further discussion on this issue.
- Culture-specific (IV or oral) antibiotics. *P. aeruginosa* infection can cause rapid deterioration of pulmonary

TABLE 1: Spirometry values of the patient.

Parameter	Predicted	Prebronchodilator (BD) measured	Pre-BD % predicted	Post-BD measured	Post-BD % predicted	% change
FVC (L)	3.57	2.96	83	3.38	95	14
FEV1 (L)	2.76	1.41	51	1.74	63	12
FEV1/FVC	77	Actual value	48		46	
FEF 25–75% (L/sec)	2.67	0.31	11	0.27	10	–12
MVV (L)	110	55	50			

(FVC: forced vital capacity; FEV1: forced expiratory volume in 1 second; FEF: forced expiratory flow; MVV: maximal voluntary ventilation).

**Fig. 1:** Different positions for postural drainage.

function. The role of inhaled antibiotics is not well-established.

- Bronchodilators in individual cases.
- Inhalational corticosteroid therapy is hypothesized to benefit the inflammatory component of bronchiectasis.
- Mucolytic agents for patients with inspissated secretions: The role of acetylcysteine as part of preoperative therapy is controversial; though it thins secretions, it does not appear to improve clinical endpoints.
- Systemic and topical hydration plays a very important role.
- Preoperative physiotherapy—postural chest drainage, percussion, vibration.
- Intravenous immunoglobulin (IVIg) for hypogammaglobulinemia.
- Vaccination schedule as in COPD patients, e.g., influenza vaccine, pneumococcal vaccine.

Q. Describe the role of preoperative physiotherapy.

Ans. Preoperative chest physiotherapy has been shown to increase the amount of expectorated sputum and benefit patients who produce over 20–30 mL of mucus daily, although there is no significant effect on FEV1.

Bronchiectasis is associated with damage of bronchial structure as a result of which there is retention and stagnation of secretions, which get infected subsequently. Bronchiectasis in the lower lobes of the lung leads to the formation of a pool of infected secretions, which can spill over and infect the other healthy parts of the lungs. The aim of postural drainage is to empty this pool of secretions by gravity-assisted drainage. The secretions are drained into the larger airways from where they can be coughed out by the patient. There are various positions for postural drainage depending on the site of the affected bronchus shown in **Figure 1**.

Q. At what stage of optimization can the patient be considered for surgery?

Ans. Patients can be considered for surgery if:

- Unilateral or localized disease
- The volume of sputum decreases to <40 mL/day
- The secretions are no longer purulent
- The secretions are not foul-smelling.

Q. What premedication would you like for this patient?

Ans. Anticholinergics cause drying of secretions and are best avoided. Sedatives and respiratory depressants should be used cautiously as loss of cough reflex might lead to

aspiration of infected contents from affected lobe to other healthy lobe/lung.

Q. Describe the technique of anesthesia you would use for this patient.

Ans. Preferred technique of anesthesia would be general anesthesia with thoracic epidural analgesia for perioperative analgesia. Lung isolation can be achieved with the use of either a bronchial blocker or a DLT.

Q. Why is postoperative analgesia important?

Ans. Adequate postoperative analgesia is important to allow early physiotherapy and ambulation, so that the patient can perform spirometry and deep breathing exercise to prevent basal atelectasis and also mobilize and cough out secretions effectively. Epidural analgesia will help to avoid complications of systemic opioids like cough suppression and respiratory depression.

Q. Describe the technique of general anesthesia in this patient.

Ans. It is important to avoid spillage of pus into the contralateral lung, which can occur during induction of anesthesia.

To prevent this, anesthesia can be administered to these patients in secretion retaining positions (*see Fig. 1*) so that infected secretions remain in affected lobes (lobe containing the abscess is in the most dependent position relative to the rest of the tracheobronchial tree). In this case, with a left lower lobe abscess, induction should be carried out in the left lateral semisitting position. A rapid sequence induction is important to avoid prolonged positive pressure ventilation, which can again increase chances of spillage (there is no need for cricoid pressure here as we are not trying to prevent gastric aspiration but a translobar/transpulmonary aspiration of secretions). Adequate preoxygenation is necessary to prevent hypoxemia during induction. One can intubate using a single-lumen tube followed by lung isolation with a bronchial blocker. Alternatively, lung separation can be achieved using a DLT. The airway and preoperative imaging should be carefully assessed to rule out anomalies, which can interfere with placement of a DLT or bronchial blocker. Alternatively, lung isolation can be achieved while patient is awake, especially in those situations where amount of secretion continues to be high in spite of all efforts. Bronchial blockers may be preferable because they can be used (under fiberoptic bronchoscope guidance) for selective lobe isolation when patient is awake and thus will prevent contamination of the remaining healthy lobes of the affected lung during induction. Similarly placement of DLT can be done in awake patient after giving topical anesthesia to upper airway.

While ventilating the healthy lung care should be taken to use low tidal volume to prevent volutrauma. Oxygen

supplementation via continuous positive airway pressure (CPAP) may be applied to the infected lung to decrease the shunt fraction to prevent hypoxemia.

Closed circuit gas delivery system provides humidified gases. If semiopen or open circuits are used, heat and moisture exchanger (HME) filter should be added to circuit for humidification. Adequate humidification is essential for prevention of drying of secretions. Frequent suctioning should be carried out, using separate catheters for infected and noninfected areas. Both rigid and fiberoptic bronchoscopes should be readily available for suctioning of the airway. Intraoperative sputum samples should be collected for culture and antibiotic sensitivity. Gram's stain of the sputum can guide antimicrobial therapy which can be further titrated with culture sensitivity.

Q. What monitors would you like to use for this patient?

Ans. Monitoring should include ECG, pulse oximetry, noninvasive blood pressure monitoring, capnography, urinary catheter (optional), and temperature probe. An invasive arterial line may be useful for frequent blood gas monitoring during one-lung ventilation.

Q. What is the postoperative management of this patient?

Ans. The patient can be extubated on table if she is warm, awake, and comfortable. Early physiotherapy should be instituted. Bronchodilator therapy should be continued in the immediate postoperative period.

Supplemental oxygen if required should be strictly humidified and SPO₂ should be monitored.

Q. What are postoperative complications in these patients?

Ans.

- Bronchopleural fistula and prolonged air leak
- Difficulty in weaning from ventilator
- Empyema
- Arrhythmias
- Atelectasis
- Pneumonia.

SUGGESTED READING

1. Fauci AS, Braunwald E, Kasper DL, Hauser SL, Longo DL, Jameson JL, et al. *Harrison's Principles of Internal Medicine*, 17th edition. New York: McGraw-Hill; 2008.
2. Hill AT, Sullivan AL, Chalmers JD, De Soyza A, Elborn JS, Floto RA, et al. *British Thoracic Society Guideline for bronchiectasis in adults*. *Thorax*. 2019;74:1-69.
3. McShane PJ, Naureckas ET, Tino G, Strek ME. Non-cystic fibrosis bronchiectasis. *Am J Respir Crit Care Med*. 2013;188:647-56.
4. Miller RD, Eriksson LI, Fleisher LA, Wiener-Kronish JP, Young WL. *Miller's Anesthesia*, 7th edition. Philadelphia: Churchill Livingstone; 2010.

Chronic Obstructive Pulmonary Disease

Atul Prabhakar Kulkarni, Madhavi Desai, Aparna Chatterjee

A 62-year-old, 65 kg male patient, with a periampullary carcinoma pancreas, is posted for pancreaticoduodenectomy. He presents with cough and yellowish mucoid expectoration. He can climb two flights of stairs with difficulty. He is a chronic smoker who has smoked 20 packs/day for 30 years, still smokes around 10 cigarettes a day since diagnosis. One year back, he was admitted to a nursing home with dyspnea and cough with expectoration. He was treated with salbutamol and ipratropium puffs, budesonide inhaler and was given oxygen therapy. His examination reveals grade 2 clubbing, occasional wheeze and cardiovascular system examination is unremarkable.

Q. What is chronic obstructive pulmonary disease?

Ans. Chronic obstructive pulmonary disease (COPD) is a common, preventable and treatable disease that is characterized by persistent respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases. It incorporates three disorders—(1) emphysema, (2) small airway disorder or abnormality, and (3) chronic bronchitis. The chronic airflow limitation that is characteristic of COPD is caused by a mixture of small airway disease (e.g., obstructive bronchiolitis) and parenchymal destruction (emphysema), the relative contributions of which vary from person to person, but the dominant clinical feature is airflow limitation that is not fully reversible.

The airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lung to noxious particles. Poorly reversible airflow limitation associated with bronchiectasis, cystic fibrosis and fibrosis due to tuberculosis are not included in the definition of COPD.

Chronic bronchitis is defined clinically as chronic productive cough for 3 months in each of 2 successive years in a patient; in whom other causes of productive chronic cough have been excluded.

Emphysema is defined as the presence of permanent enlargement of the airspaces distal to the terminal bronchioles, accompanied by destruction of their walls and without obvious fibrosis.

The important causes of COPD are tobacco smoking, outdoor air pollution increasing inhaled particulate

burden, occupational exposure to dusts especially in coal mining, gold mining, and the textile industry; and alpha-1 antitrypsin deficiency. In rural India, cooking on biomass fuel mainly coal fire is the most common cause of COPD in females. Marijuana smoke is also known to cause COPD.

Q. How do you diagnose COPD?

Ans. Clinical diagnosis of COPD is considered in a patient who has dyspnea, chronic cough or sputum production, and/or history of exposure to risk factor for disease. Presence of postbronchodilator forced expiratory volume in 1 second (FEV₁)/forced vital capacity (FVC) < 0.7 on spirometry; indicating persistent airflow limitation; is required to confirm the diagnosis in this clinical context.

Q. What are the mechanisms underlying airflow limitation in COPD?

Ans. The hallmark of COPD is chronic airflow limitation. This is caused by a combination of inflammation and narrowing (obstructive bronchiolitis) of the small airways and destruction of the lung parenchyma. There is loss of alveolar attachments to the small airways due to inflammation with consequent decrease in the elastic recoil of the lungs. This leads to airway collapse during early expiration and thus airflow limitation. It occurs particularly in emphysematous patients, which results in marked dyspnea on exertion.

Q. How do you differentiate between COPD and asthma?

Ans. Asthma is a disease characterized by chronic airway inflammation, reversible expiratory airflow obstruction in response to various stimuli, and bronchial hyperreactivity. Asthma is an episodic disease with acute exacerbations

interspersed with symptom-free periods. It is characterized by mucus production and bronchoconstriction (**Table 1**).

TABLE 1: Differences between chronic obstructive pulmonary disease (COPD) and asthma.

Chronic obstructive pulmonary disease	Asthma
COPD occurs later in life (middle age)	Asthma patients present early in life (often in childhood)
Associated with prolonged smoking, exposure to certain chemical dust or biomass fuel	Often a history of allergy, rhinitis and/or eczema is present
It is slowly progressive	Symptoms vary from day to day and peaking in the night or early morning
Airways as well as parenchyma are affected in COPD	Only the airways are affected in asthma
Airflow limitation in these patients is largely irreversible as it is structural in nature	Airflow limitation is largely reversible. Airway hyper-responsiveness is a hallmark of asthma
Inflammation is neutrophilic and CD8-driven in COPD; therefore, inhaled steroids are largely ineffective	Inflammation is predominantly eosinophilic and CD-4 driven; therefore, inhaled steroids are effective

(COPD: chronic obstructive pulmonary disease).

Q. How is dyspnea graded in COPD patients?

Ans. According to the modified Medical Research Council (mMRC) questionnaire, this patient has grade 1 dyspnea. The mMRC grades dyspnea is shown in **Table 2**.

TABLE 2: The modified Medical Research Council grading for dyspnea.

Description	Grade	Degree
Only gets breathless on strenuous exercise	0	None
Shortness of breath when hurrying on the level/walking up slight hill	1	Mild
Walks more slowly than people of the same age on the level because of breathlessness or has to stop for breath walking at own pace on level	2	Moderate
Stops for breath after walking about 100 m after few minutes on the level	3	Severe
Too breathlessness to leave the house, to dress and undress	4	Very severe

Q. How will you investigate this patient? When will you ask for echocardiographic evaluation of a COPD patients?

Ans. A patient with COPD commonly has various extrapulmonary comorbidities, such as nutritional abnormalities resulting in weight loss and skeletal muscle dysfunction. They are at an increased risk of myocardial infarction, angina, osteoporosis, bone fractures, respiratory infection, depression, diabetes, sleep disorders, anemia

and glaucoma. The long-standing COPD ultimately results in pulmonary hypertension and right ventricular (RV) hypertrophy leading to cor pulmonale. The investigations are directed at detecting presence of these comorbidities, which will help in preoperative optimization of the patient.

Complete blood count may reveal either anemia (nutritional deficiency) or high hemoglobin [carboxyhemoglobin (COHb) may be present] and raised white cell count in presence of active infection.

Depending on severity and duration of COPD, chest X-ray may show flattening of diaphragm, horizontal ribs and hyperinflated lungs and tear drop or tubular heart. Look for bullae in X-ray chest or computed tomography (CT) chest if available. Many patients with moderate or severe COPD develop cystic airspaces in the lung parenchyma known as bullae. These bullae will often be asymptomatic unless they occupy >50% of the hemithorax, in which case the patient will present with findings of restrictive respiratory disease in addition to their obstructive disease.

Spirometry frequently reveals reduced FEV1 and FEV1/FVC ratio with minimal or no improvement after bronchodilators.

Electrocardiogram may show signs of RV hypertrophy with right axis deviation and/or and right atrial hypertrophy and dilatation (P pulmonale).

Nocturnal desaturation and frequent exacerbations of COPD lead to chronic hypoxia, which can result in RV dysfunction. In addition to increasing severity of COPD, chronic hypoxia causing polycythemia together contribute to pulmonary hypertension (pulmonary artery pressure > 20 mm Hg) and RV dysfunction. Echocardiography will be indicated in patients with COPD who have fatigue, dyspnea that is disproportionate to the severity of COPD and history of paroxysmal nocturnal dyspnea. RV dysfunction is present in almost 50% of the patients with COPD which is developed due to recurrent episodes of hypoxia. A two-dimensional (2D) echocardiography will reveal increased pulmonary artery pressures, dilation and/or hypertrophy of right ventricle. Left ventricular dysfunction may be evident if associated ischemic heart disease is present.

Arterial blood gas (ABG) analysis will show a normal or near normal pH, borderline PO₂ and normal or raised CO₂ and HCO₃.

Q. How is COPD classified/staged?

Ans. Assessment of the airflow limitation severity in COPD is on the basis of the FEV1% of predicted values. Classification of COPD based on postbronchodilator FEV1 in whom FEV1/FVC is <0.70. Spirometry should be performed after the administration of an adequate dose of at least one short-acting inhaled bronchodilator in order to minimize variability.

Spirometric classification of the Global Initiative for Chronic Obstructive Lung Disease (GOLD):

GOLD COPD severity grading is shown in **Table 3**.

TABLE 3: The GOLD COPD severity grading.

Stage	Severity	Spirometry	
		FEV1/FVC*	FEV1% predicted
GOLD I	Mild	<0.70	>80
GOLD II	Moderate	<0.70	50–80
GOLD III	Severe	<0.70	30–50
GOLD IV	Very severe	<0.70	<30

*Postbronchodilator

The American Thoracic Society classification of COPD:

- Stage I: >50% predicted
- Stage II: 35–50% predicted
- Stage III: <35% predicted.

As per this criteria, stage I patients should not have significant dyspnea, hypoxemia or hypercarbia and other causes should be considered if these are present.

Q. Can we predict postoperative pulmonary complications (POPCs)? When is patient deemed unfit for anesthesia and surgery?

Ans. Unlike in pulmonary resection, there is no cut-off value of FEV1 or any other spirometric index to consider these patients unsuitable for surgery. Many studies have attempted to predict POPC, though not specifically in COPD patients.

The utility of spirometry and blood gas analysis in predicting risk of POPC is questionable. Various studies and systematic reviews have shown that age > 65 years, smoking 40 packs/year or more, FEV1 < 1 L/min, FVC < 1.5 L/min, PO₂ < 75 mm Hg and PCO₂ > 45 mm Hg indicate increased risk of POPCs. But the most significant factors are abnormal clinical findings on pulmonary examination, presence of preoperative respiratory symptoms (cough, dyspnea, excessive sputum production, chest pain and wheezing) and type of surgery (upper abdominal). Different authors suggest different criteria to predict risks of POPCs. Commonly used are:

- *Nunn and Milledge criteria:*
 - FEV < 1 L, normal arterial partial pressure of oxygen (PaO₂) and arterial partial pressure of carbon dioxide (PaCO₂): Low risk of POPC
 - FEV1 < 1 L, low PaO₂ and normal PaCO₂: Patient will need prolonged O₂ supplementation
 - FEV1 < 1 L, low PaO₂, and high PaCO₂: Patient may need postoperative ventilation.
- *Based on spirometry:*
 - Predicted FVC < 50%
 - Predicted FEV1 < 50% or < 2 L

- Predicted maximum voluntary ventilation (MVV) < 50% or < 50 L/min
- Predicted diffusing capacity of lung for carbon monoxide (DLCO) < 50%
- Predicted RV/total lung capacity (TLC) > 50%.

• *Based on patient criteria and type of surgery:*

- *Patient-dependent factors:* Current smoker, reduced health status [the American Society of Anesthesiologists (ASA) grade >2], old age (>70 years in COPD patients), COPD with exercise intolerance
- *Surgery-dependent factors:* Abdominal surgery (open > minimal invasive), thoracic surgery, long duration of anesthesia (>4 hours), general anesthesia (GA) [vs. regional anesthesia (RA)].

Also it is necessary to keep in mind that these patients are also more prone to develop exacerbation of RV dysfunction and failure due to intraoperative events, such as acidosis, hypercarbia, hypoxia and fluid overload.

A recent study found that these patients were more likely to develop POPC if they:

- Underwent upper abdominal (vs. lower abdominal) surgery
- Had emergency (vs. elective) surgery
- Had nasogastric tube present
- Underwent reoperation during same admission.

Q. Describe the pulmonary function tests (PFTs) commonly done at the bedside and their implications.

Ans.

Cough test: It is performed by asking the patient to take a deep inspiration and cough once. Test is positive if the first cough leads to recurrent coughing, which is suggestive of underlying bronchitis.

Wheeze test: The patient is asked to take five deep inspirations/expiration; the patient is then auscultated between the shoulder blades posteriorly to determine the presence or absence of wheezing.

Maximum laryngeal height: The distance between the top of the thyroid cartilage and the suprasternal notch at the end of expiration. If it is less than 4 cm, it is abnormal. It is an accurate sign of obstructive airway disease compared to PFTs.

Forced expiratory time (FET): Place the bell of the stethoscope over the trachea in the suprasternal notch and set the stopwatch to zero. Instruct the patient to take in the deepest breath possible and then to blow it all out as fast as possible. Start the stopwatch as the patient begins to exhale, stop it as soon as audible expiration is no longer heard. An FET > 6 seconds indicates severe expiratory airflow obstruction with %FEV1 < 50%. Three trials are done and the

results are averaged. This clinically measured FET correlates well with the FET measured by spirometry.

Sabrasez breath-holding time (BHT): Ask patient to take a deep breath and hold his breath as long as possible. Place a stethoscope over the trachea to identify early expiration. A BHT > 40 seconds is normal. A BHT between 20 and 30 seconds indicates compromised cardiopulmonary reserve. BHT < 20 seconds indicates very poor cardiopulmonary reserve.

Snider's match test: The test is used to measure the patient's maximum breathing capacity (MBC). Place a lighted match stick at varying distances from the patient's mouth. Instruct the patient to sit, keep his mouth open and blow (without pursing the lips) the candle off. Ability to blow the candle off at 22 cm from the mouth indicates MBC > 150 L/min (MBC < 100 L/min at 15 cm and MBC < 50 L/min at 7.5 cm). Patients with moderate-to-severe COPD have great difficulty with this test. Some patients require multiple trials to huff out the match at 15 cm, and some with severe disease are unable to accomplish this. Need for use of therapeutic oxygen in patients with COPD is a contraindication to this test.

Single breath count test: The patient is asked to count out loud numbers from 1 onward after a maximal inspiration. Individuals with normal respiratory function can count to 50 or more. A single breath count of <15 indicates severe impairment of vital capacity (VC).

Peak expiratory flow (PEF) rate measurement: The use of PEF rate measurements has been advocated in the management of asthma but has no utility in managing COPD. In COPD, there is a poor relationship between PEF and FEV₁, and it is impossible to predict FEV₁ from the PEF or vice versa. PEF may underestimate the degree of airway obstruction in COPD and is relatively insensitive to obstruction of the small airways (mild or early obstruction). Moreover, PEF is very dependent on patient effort.

Q. Discuss the effects of smoking on various systems.

Ans. Cigarette smoke consists of a gaseous phase (80–90%) and a particulate phase. Gaseous phase consists of mainly nitrogen, oxygen, carbon dioxide, carcinogens (hydrocyanic acid and hydrazine), ciliotoxins, irritants (hydrocyanic acid), carbon monoxide (CO) and some others. Nicotine is the main component of particulate phase.

Cardiovascular system: Nicotine stimulates the adrenal medulla to secrete adrenaline, resets the carotid body and aortic receptors to maintain a higher blood pressure, and stimulates autonomic ganglia, increasing sympathetic tone. The result is an increase in systolic and diastolic blood pressure, heart rate, and peripheral vascular

resistance; leading to an increase in oxygen demand by the myocardium. An increase in coronary vascular resistance decreases the myocardial oxygen supply-demand ratio. The half-life of nicotine is 30–60 minutes. Following the smoking of one cigarette, the pressor response lasts for about 30 minutes. Three to four hours of abstinence results in insignificant side effects due to nicotine and a significant improvement of the myocardial oxygen supply-demand ratio.

Effects of COHb: Cigarette smoke contains 400 parts per million (ppm) CO. In the blood, CO combines with hemoglobin to form COHb. In smokers, the amount of COHb in the blood ranges from 5 to 15%. The affinity of CO for hemoglobin is 200 times that of oxygen. It shifts the oxyhemoglobin curve to the left due to: (1) its high affinity for Hb, (2) a change in shape of the oxyhemoglobin curve from a sigmoidal to a more hyperbolic curve by COHb, and (3) depletion of 2,3-diphosphoglycerate by CO. These mechanisms lead to chronic tissue hypoxia. CO by itself may cause arrhythmias. The half-life of COHb depends clearly on pulmonary ventilation: at rest 4–6 hours, with strenuous exercise 1 hour, during sleep 10–12 hours. With 100% oxygen, the half-life is reduced to 40–80 minutes. Thus, on advising patients before anesthesia, these variations should be noted. During the daytime, abstinence for 12 hours is sufficient to get rid of CO. If an operation is scheduled for the next morning, the patient should not smoke the previous evening.

Hemostatic system: Smoking increases the production of hemoglobin, red blood cells, white blood cells, and platelets, fibrinogen and increases platelet reactivity. This results in an increase in the hematocrit and the blood viscosity, leading to an increased thrombotic tendency. Therefore, incidence of arterial thromboembolic disease is more in smokers; however, incidence of deep vein thrombosis (DVT) has not been shown to be increased. Chronic hypoxia to the cardiac muscle and the increase in incidence of thromboembolic disease causes smokers to be at a 70% greater risk of coronary artery disease compared with nonsmokers, and the postoperative mortality in smokers is higher than in nonsmokers.

Respiratory system: Irritants in smoke increase mucus secretions. The mucus becomes hyperviscous. Cilia become inactive and are destroyed by ciliotoxins, which lead to decreased tracheobronchial clearance. Cigarette smoke disrupts the epithelial lining of the lung, causing an increase in pulmonary epithelial permeability. This loss of epithelial integrity allows irritants to penetrate the epithelium more easily and stimulate the subepithelial irritant receptors, resulting in increased reactivity. Smoking leads to small

airway narrowing, causing an increased closing volume. Pulmonary surfactant is also decreased. These lead to small airway disease. An increase in pulmonary proteolytic enzymes or elastolytic enzymes causes loss of elastic lung recoil and emphysema. 25% of smokers suffer from chronic bronchitis.

Gastrointestinal system: Smoking makes the gastroesophageal sphincter incompetent, which allows reflux, with accompanying risks of pulmonary aspiration. The incompetence in the gastroesophageal sphincter begins within 4 minutes of beginning to smoke and returns to normal within 8 minutes after the end of smoking.

Q. Discuss the preoperative measures undertaken to optimize a COPD patient.

Ans. Cessation of smoking is the single most important intervention in the preoperative period. A study by Warner found that the incidence of POPCs was highest (57%) in patients who quit smoking for <8 weeks; it decreased to 14.5% if cessation was >8 weeks. It was least in (11.9%) in those who never smoked. Current smokers (33%) also had a high risk of postoperative complications. It is hypothesized that the absence of the irritant effect of cigarette smoke in the postoperative period inhibits coughing and leads to retention of secretions and small airway obstruction, in patients who stopped smoking < 8 weeks. Within 12 hours of cessation, the COHb levels drop. Long-term cessation (>8 weeks) leads to improvement in mucociliary function with increased sputum clearance and also reduced airway reactivity and sputum production. The risk for patients who quit > 6 months is the same as those who never smoked. Smoking has also been shown to delay wound healing.

Treat chest infection with appropriate antibiotics.

Stable patient with COPD usually receives a combination therapy with inhaled long-acting beta-2 agonists (LABAs) like salmeterol, formoterol, vilanterol and long-acting antimuscarinic agent (LAMA) like glycopyrronium bromide, or a combination of short-acting beta-2 agonists (SABAs) and short-acting antimuscarinic agent (SAMA) like ipratropium bromide. Commonly used is salbutamol and ipratropium in inhaled form. These need to be continued in perioperative period as scheduled.

Steroids: Inhaled corticosteroids (ICSs) improve symptoms, lung function; and reduce exacerbations when FEV1 is <60% of predicted value, but have shown to increase incidence of oral candidiasis.

Many of these patients might be receiving steroids. Recommendations for perioperative management of patients on steroid are given in **Table 4**.

Patients with cor pulmonale may be receiving diuretics and/or digitalis. These should be continued perioperatively.

Hydration and mucolytic therapy will help in loosening of secretions and along with chest physiotherapy will help in clearing the chest.

TABLE 4: Recommendations for steroids in perioperative patient.

Dose	Surgery	Recommended dose
<10 mg/day	Minor/ moderate/ major	Additional steroid cover not required [assume normal hypothalamic-pituitary-adrenal (HPA) response]
>10 mg/day	Minor	25 mg of hydrocortisone at induction and normal medications postoperative
>10 mg/day	Moderate	Usual dose preoperative and 25 mg hydrocortisone IV at induction then 25 mg IV TDS for 1 day then recommence preoperative dosage
>10 mg/day	Major	Usual dose preoperative and 100 mg hydrocortisone at induction then 100 mg IV TDS for 2–3 days

Pulmonary rehabilitation improves health status and effort tolerance in COPD patients.

Preoperative *incentive spirometry* has always been recommended for reducing POPCs, though there is no supportive evidence.

Q. How will you premedicate this patient?

Ans. Anxiolysis is important in COPD patients. In absence of hypercapnia on a baseline ABG, it is safe to premedicate these patients with a small dose of benzodiazepine as it is unlikely to produce central depression. Benzodiazepines do not alter bronchial tone. In other patients, it may be safer to administer premedication in the preoperative holding area in monitored environment. There is no role of routine anticholinergic premedication as most of modern anesthetic agents have minimum respiratory irritant properties as compared to the old ones.

Q. What anesthetic technique would you choose in this patient? Why?

Ans. Regional anesthesia has the advantage of avoiding airway instrumentation and bronchospasm. The disadvantages of a regional block include inadequate muscle relaxation, coughing, bucking, need for high levels of spinal or epidural block and increased parasympathetic tone, which may cause bronchospasm. RA can decrease expiratory reserve volume (ERV) by ~50%, which may be detrimental if the patient is dependent on active expiration. Hypotension due to RA, patient discomfort due to prolonged procedure and shivering, need for heavy sedation with RA (which may be worse than light GA) are other considerations. Also it is important to remember that hypoxemia or hypercarbia that can develop in high levels of motor blockade may adversely affect RV function.

General anesthesia offers the advantages of control of ventilation with good muscle relaxation. This ensures oxygenation and CO_2 elimination and overcomes the decrease in lung compliance, increased resistance and decreased functional residual capacity (FRC).

A combination of GA with regional analgesia would be ideal in our patient. The regional analgesia can be achieved by continuous thoracic epidural analgesia. Apart from all benefits of GA, epidural anesthesia (EA) adds the benefit of excellent analgesia, reduced requirement of muscle relaxants and opioids. Added advantages include postoperative analgesia that would avoid use of narcotics, facilitation of early ambulation, better performance of respiratory therapy maneuvers and possible reduction in POPCs and deep venous thrombosis. Rectus sheath block or subcostal transversus abdominis plane block with continuous catheter technique can be the alternatives to the epidural analgesia in case the thoracic epidural is not possible.

Q. What monitoring would you institute in this patient?

Ans. The standard monitoring will include electrocardiogram, heart rate, end-tidal CO_2 , pulse oximetry, temperature, invasive blood pressure monitoring (in view of major surgery with fluid shifts) and hourly urine output monitoring. Invasive arterial pressure monitoring offers the advantage of beat-to-beat pressure monitoring as well as sampling for ABG analysis. Central venous monitoring will help in optimizing fluid therapy.

Q. What are the options for induction of anesthesia?

Ans. It is best to avoid thiopentone as thiobarbiturates may cause histamine release. If barbiturates have to be used, it is preferable to use oxybarbiturates (methohexitone). It is important to remember that airway instrumentation or stimulation under light thiopentone anesthesia may provoke a bronchospasm. In cardiostable patients, propofol would be agent of choice for anesthesia induction as it offers marked protection against bronchospasm; however, hypotension needs to be guarded against. Though many anesthesiologists prefer ketamine for its bronchodilator effect, it is dose related and not predictable. In addition, it can cause tachycardia and hypertension and an increase in pulmonary vascular resistance via indirect adrenergic stimulation. It also leads to an increase in airway secretions.

Q. How will you secure the airway and conduct anesthesia?

Ans. Laryngeal mask airway (LMA) has been associated with a lower incidence of bronchospasm due to less airway stimulation; we obviously cannot use it in the present case as this patient is undergoing upper abdominal surgery and may need postoperative ventilation. Also the lung

compliance may be poor needing high airway pressure for ventilation, which may be a problem with LMA. This is because airway pressures $> 30 \text{ cm H}_2\text{O}$ (with 2nd-generation LMAs) are known to be associated with gastric insufflation; which may lead to regurgitation and aspiration of gastric contents.

It is best to use a nondepolarizing muscle relaxant (NDMR), narcotic and adequate plane of anesthesia before intubating the patient. It is known that NDMRs, which affect the M_2 (muscarinic type 2) receptors more than the M_3 receptors cause and enhance bronchoconstriction. These include gallamine, pipecuronium, which are not in much use now. Vecuronium, rocuronium, and pancuronium affect M_3 receptors and can be used safely. Benzyl quinolones like atracurium, mivacurium cause dose-dependent histamine release and increased bronchial tone and should be avoided. This histamine release is not seen with cisatracurium and it can be used.

Opioids induce cough suppression and are one of the components of balanced anesthesia. Fentanyl in large doses, alfentanil and sufentanil can cause chest rigidity, mimicking bronchospasm. This effect is common with faster injection and increasing age of the patient. Morphine and pethidine are known to cause histamine release.

Among the inhalational agents, halothane is the most potent bronchodilator. It suppresses airway reflexes, causes direct relaxation of airway smooth muscle in a dose-related manner due to beta-adrenergic receptor stimulation, which is decreased by beta-blocking agents. Sevoflurane and isoflurane at higher minimum alveolar concentration (MAC) are equally good, though isoflurane due to its irritant smell can provoke bronchospasm.

Administration of topical lignocaine for intubation or before endotracheal suctioning is no more recommended as it itself can provoke bronchospasm.

Q. How will you diagnose and treat intraoperative bronchospasm?

Ans. Increased peak and plateau airway pressures, decreasing slope of expired CO_2 and wheeze or absence of breath sounds are signs suggestive of intraoperative bronchospasm. This may lead to hypoxemia, hypercarbia and hypotension due to dynamic hyperinflation and development of autospontaneous end-expiratory pressure (auto-PEEP) [intrinsic PEEP (PEEPi)]. Causes of intraoperative increase in peak airway pressure are:

- Light anesthesia, coughing, bucking
- Obstruction in the circuit
- Blocked/kinked tube
- Endobronchial intubation
- Bronchospasm
- Pneumothorax

- Major atelectasis
- Pulmonary edema
- Aspiration pneumonia
- Head-down position, bowel packing.

On noticing raised airway pressures, rule out various causes mentioned above. Once bronchospasm is diagnosed, increase the FiO_2 to 1.0. As the most common cause of bronchospasm is surgical stimulation under light anesthesia, deepen plane of anesthesia. Perform thorough endotracheal suctioning after ensuring adequate depth of anesthesia. The pharmacological options to treat bronchospasm are use of inhaled [or nebulized, or metered-dose inhaler (MDI)] beta-2 agonists, use either nebulized beta-2-agonists, or in severe case use subcutaneous terbutaline or intravenous adrenaline. Inhaled terbutaline and albuterol have similar efficacy via the intravenous route. Often more than two puffs may be needed to counter the acute bronchospasm. Intravenous MgSO_4 (1–2 g over 20 minutes) can also be used. Intravenous corticosteroids are indicated in severe bronchospasm. Aminophylline is no more recommended because of resistant arrhythmias associated with the drug.

Q. What is auto-PEEP? What is pathogenesis of auto-PEEP? What are other situations that can lead to generation of auto-PEEP?

Ans. In a patient with COPD, during a stage of bronchospasm, increased bronchial smooth muscle tone leads to resistance to air movement. Since inspiration is an active process, this phase is not much affected; however, expiration being a passive process that totally depends on caliber of airway and elastic recoil of lungs gets prolonged. If next breath is delivered to the lungs (spontaneous or controlled) before complete exhalation of previous breath, there will be accumulation of air in alveoli (breath stacking). This “trapped air” generates positive airway pressure at the end of expiration, and this is known as “auto-PEEP” (self-generated PEEP) or “PEEPi”. This cumulative increase leads to increasing amount of air being trapped in the lungs and results in over expansion of lungs leading to “dynamic hyperinflation” of the lungs. Other causes of auto-PEEP are mucus plugging of the airways, large minute ventilation, block in the expiratory circuit of the ventilator [such as blocked heat moisture exchange (HME) filter] and ventilating with small endotracheal tube with inadequate expiratory time that leads to incomplete expiration.

Q. How do you confirm development of auto-PEEP? How do you treat it?

Ans. Most modern ventilators will allow direct measurement of auto-PEEP. Expiratory flow not reaching zero in flow-time graph (Fig. 1) or flow-volume loop (Fig. 2) reveals incomplete expiration resulting in air trapping and auto-PEEP.

For intraoperative diagnosis, apnea test can be carried. Hypotension, if it develops over time, is almost always due to excessive hyperinflation and auto-PEEP in the absence of hypovolemia. Disconnect the patient from the ventilator for 30 seconds (apnea). If this leads to normalization of blood pressure (due to improved expiration and thus venous return), this confirms the diagnosis of auto-PEEP. Auto-PEEP can be eliminated by increasing expiratory time; this can be achieved with reduction in tidal volume, respiratory rate, and increasing the peak inspiratory flow rate.

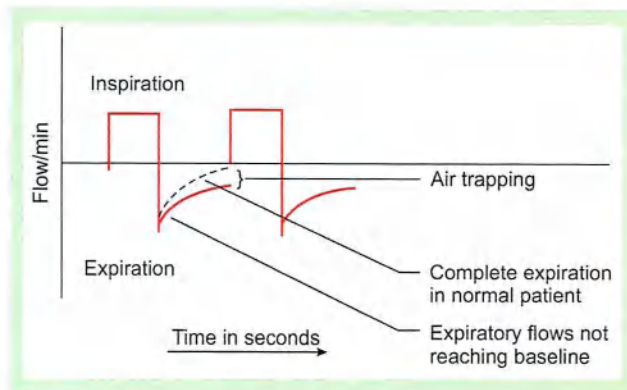


Fig. 1: Flow-time curve.

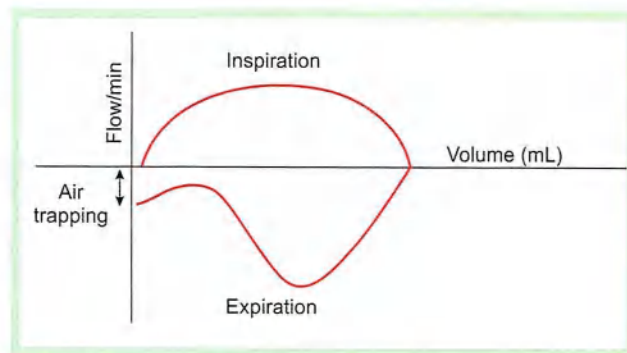


Fig. 2: Flow-volume loop.

Q. Do you anticipate any problems during reversal? When will you extubate this patient?

Ans. Reversal of muscle relaxation with neostigmine may provoke bronchospasm; this can be countered with administration of atropine 1.2–1.8 mg or glycopyrrolate 0.6 mg before neostigmine. Extubation under deeper plane of anesthesia may reduce incidence of bronchospasm, in this case delayed extubation may be preferred. Actually this patient may require a period of postoperative ventilation due to prolonged surgery and hypothermia. The patient can then be extubated once fully awake and obeying commands, has sustained head lift and adequate gas exchange.

Q. Discuss postoperative management of this patient.

Ans. Postoperative analgesia plays a pivotal role in the outcome of these patients. Continued utilization of the epidural infusion (local anesthetic alone or combined with small dose of narcotic) will ensure adequate analgesia. The only large randomized prospective study of epidural versus systemic analgesia was the MASTER trial, mainly for upper abdominal surgeries. Postoperative respiratory failure was significantly reduced in the epidural group (23% vs. 30%) with no differences in other types of complications or mortality. This beneficial effect of thoracic epidural analgesia seems to be most pronounced in patients with underlying lung disease such as COPD. This may be further improved upon by addition of systemic nonsteroidal anti-inflammatory drugs (NSAIDs) and/or paracetamol. Adequate analgesia will allow administration of postoperative respiratory therapy in the form of incentive spirometry and deep breathing exercise, will prevent development of basal atelectasis, and improve gas exchange. Deep venous thrombosis prophylaxis with either unfractionated or low-molecular-weight heparin should be given. Early mobilization and enteral feeding (via nasojejun tube or jejunostomy) are also important.

Q. Enumerate likely POPCs in this patient.

Ans.

- **Decreased FRC:** The large incision causes decrease in FRC due to postoperative pain, splinting of the diaphragm and conversion from diaphragmatic to rib cage breathing pattern, decreased sputum clearance.
- **Bibasilar atelectasis** with retained secretions can lead to postoperative pneumonia. This can be prevented with incentive spirometry, chest physiotherapy, and early ambulation.
- **Acute exacerbation of COPD:** Severity of exacerbation is classified as follows based on clinical symptoms and signs (**Table 5**).
- Methylxanthines are not recommended because of their very narrow therapeutic index and high side effects profile; especially intractable arrhythmias.
- Inhaled short-acting bronchodilator therapy and systemic steroids need to be added. Antibiotics are indicated for 5–7 days to control the infection. This being abdominal surgery, patient may need intubation if the patient develops exacerbation with acute respiratory failure in immediate postoperative period. Otherwise, noninvasive ventilation is preferred.
- Delayed ambulation.
- **Deep venous thrombosis, pulmonary embolism (PE):** Poor preoperative mobility, development of polycythemia and intraoperative hypothermia make these patients particularly vulnerable to development of deep venous

thrombosis. Ideally such patients should receive one dose of low-molecular-weight heparin preoperatively. In the presence of absolute or relative contraindications, mechanical prophylaxis (in the form of graduated compression stockings and/or sequential calf compression devices) should be resorted to intraoperatively as well. As soon as possible postoperatively, DVT prophylaxis should be commenced.

- Right ventricular dysfunction/failure.

TABLE 5: Classification of acute exacerbation of COPD based on clinical signs and symptoms.

	Acute exacerbation with no respiratory failure	Acute exacerbation with respiratory failure—not life-threatening	Acute exacerbation with respiratory failure—life-threatening
Respiratory rate/minute	20–30	>30	>30
Accessory muscles	Not used	used	used
Mental status	No change	No change	Acute change
Hypoxemia	Improves with supplemental oxygen; FiO_2 0.28–0.35	Improves with supplemental oxygen; FiO_2 0.25–0.3	Needs FiO_2 > 0.4
Hypercarbia	No increase from baseline PaCO_2	Increase from baseline PaCO_2 or elevated to 50–60 mm Hg	PaCO_2 elevated to >60 mm Hg or associated with respiratory acidosis; pH < 7.25

(COPD: chronic obstructive pulmonary disease; FiO_2 : fraction of inspired oxygen; PaCO_2 : arterial partial pressure of carbon dioxide).

Q. Discuss the causes and management of hypoxia in a postoperative patient.

Ans. Hypoxia is the most common postoperative complication. Use of pulse oximetry has improved the detection and management of postoperative hypoxia. In elderly patients, those with shivering or hemodynamic instability, the degree of hypoxia is increased manifold.

Arterial hypoxemia is defined as $\text{PaO}_2 < 55$ mm Hg or $\text{SaO}_2 < 88\%$ or $\text{PaO}_2 > 55$ —<60 mm Hg with right heart failure or erythrocytosis.

The common causes of postoperative hypoxia are:

- **Diffusion hypoxia:** Nitrous oxide (N_2O) is much more soluble (>40 times) in blood than nitrogen. Once N_2O is discontinued at the end of anesthesia, it diffuses out into the alveoli. If the patient is breathing air at this time, FiO_2 in the alveoli is <0.21, thus reducing alveolar PO_2 . This can be prevented by giving 100 O_2 by mask for at least 5 minutes after discontinuation of N_2O .

- *Low inspired concentration of oxygen:* This may occur in rare instances, such as crossing of N₂O and O₂ pipelines during hospital construction, low barometric pressure, switching off of adapters, nitrogen flooding of the gas pipeline during repairs.
- *Alveolar hypoventilation:*
 - *Residual effect of neuromuscular blockers (NMBs) opiates, pain*—use analgesia, adequate reversal of NMB, administration of supplemental O₂
 - *Air way obstruction:* Insertion of oro- or nasopharyngeal airways, supplemental O₂
 - *Bronchospasm:* Treatment of cause and supplemental O₂.
- *Intrapulmonary right-to-left shunt:* It is the most common cause of postoperative hypoxia:
 - Atelectasis (collapse of part or whole lung) can occur due to a variety of etiologies in the postoperative period:
 - Bronchial obstruction because of secretions or blood
 - Endobronchial intubation with collapse of the opposite lung
 - Pneumothorax
 - Diffuse airway collapse. When the closing capacity exceeds FRC, airways collapse during tidal ventilation resulting in right-to-left shunting.
- *Pulmonary edema:* It can occur due to left ventricular failure as a result of sudden increase in afterload (commonly seen as increase in the systolic blood pressure) in the postoperative period. Pulmonary edema of the noncardiac origin [acute respiratory distress syndrome (ARDS)] due to increased pulmonary capillary permeability may occur in aspiration, postsurgical sepsis, trauma, and acute severe pancreatitis. Treatment will depend on cause and ventilation with high FiO₂ and PEEP, diuresis, vasopressors and inotropes (in case of shock) may be needed.
- *Pulmonary embolism:* It is rare but may be life-threatening in the immediate postoperative period. Accurate diagnosis is essential. Bedside 2D echocardiography, pulmonary angiography or spiral CT scan are the diagnostic modalities. Anticoagulation with continuous intravenous infusion of heparin and/or surgical embolectomy are the treatment options. Thrombolysis may be contraindicated in most of the postoperative cases but may be given weighing the risks and benefit in surface surgeries.

Q. Discuss oxygen therapy in COPD patients.

Ans. Regardless of whether patient has COPD or not, all hypoxic patients must be given oxygen. The goal of

supplemental oxygen is to maintain a PaO₂ 50–55 mm Hg. COPD patients often remain stable even in presence of what would be alarmingly low PO₂ levels for normal patients. Hypoxia in COPD patients can lead to a sudden cardiorespiratory arrest. It is difficult to predict the PO₂ level, which can lead to catastrophe but most COPD patients will remain oxygenated if PO₂ is <50 mm Hg. Episodes of acute exacerbation will cause further drop in the PO₂ levels. It is difficult to judge beforehand the PO₂ levels that may be achieved in COPD patients for a particular FiO₂. Hypercarbia may occur due to respiratory depression with oxygen therapy. As long as one monitors the PCO₂ and in particular pH, so as to intervene (generally at pH < 7.25), patient can be safely given low FiO₂ with a Venturi mask (0.24–0.36) and kept under observation.

SUGGESTED READING

1. Buist AS. Similarities and differences between asthma and chronic obstructive pulmonary disease: treatment and early outcomes. *Eur Respir J*. 2003;21(39):30s-5s.
2. Fisher BW, Majumdar SR, McAlister FA. Predicting pulmonary complications after nonthoracic surgery: a systematic review of blinded studies. *Am J Med*. 2002;112:219-25.
3. Global Initiative for Chronic Obstructive Lung Disease. Pocket Guide to COPD Diagnosis, Management, and Prevention: A Guide for Health Care Professionals. 2019.
4. Kanat F, Golcuk A, Teke T, Golcuk M. Risk factors for postoperative pulmonary complications in upper abdominal surgery. *ANZ J Surg*. 2007;77:135-41.
5. McAlister FA, Khan NA, Straus SE, Papaioakim M, Fisher BW, Majumdar SR, et al. Accuracy of the preoperative assessment in predicting pulmonary risk after nonthoracic surgery. *Am J Respir Crit Care Med*. 2003;167:741-4.
6. Rodrigo C. The effects of cigarette smoking on anesthesia. *Anesth Prog*. 2000;47:143-50.
7. Savas JE, Litwack R, Davis K, Miller TA. Regional anesthesia as an alternative to general anesthesia for abdominal surgery in patients with severe pulmonary impairment. *Am J Surg*. 2004;188:603-5.
8. Slinger P. Don't make things worse with your ventilator settings: How you manage the lungs during the perioperative period affects postoperative outcomes. IARS 2013 Review Course Lectures. USA: IARS; 2013.
9. Smetana GW, Lawrence VA, Cornell JE, American College of Physicians. Preoperative pulmonary risk stratification for noncardiothoracic surgery: systematic review for the American College of Physicians. *Ann Intern Med*. 2006;144:581-95.
10. Smith PR, Baig MA, Brito V, Bader F, Bergman MI, Alfonso A. Postoperative pulmonary complications after laparotomy. *Respiration*. 2010;80:269-74.
11. Straus SE, McAlister FA, Sackett DL, Deeks JJ; for the CARE-COAD1 Group. The accuracy of patient history, wheezing, and laryngeal measurements in diagnosing obstructive airway disease. *JAMA*. 2000;283:1853-7.

Intercostal Drain

Priya Ranganathan, Bhakti Trivedi, Sudivya Sharma, Madhavi Shetmahajan

A 55-year-old male patient presented with history of dull left-sided chest pain and progressively increasing breathlessness since 3 months. He gives history of cough with occasional hemoptysis, low-grade fever, and weight loss. On examination, he is malnourished. There is generalized lymphadenopathy. Examination of the respiratory system reveals grossly decreased air entry over the left hemithorax. The patient is breathless at rest and cannot walk more than a few steps. Chest X-ray and ultrasonography of the chest reveal a massive left pleural effusion. This patient is posted for cervical lymph node biopsy under general anesthesia.

Q. How would you manage this patient's pleural effusion?

Ans. Since this patient has a large pleural effusion and is symptomatic, he needs drainage of the effusion by inserting a chest tube or intercostal chest drain (ICD).

Q. What are the common indications for chest drain insertion?

Ans. Asymptomatic patients with a minimal pneumothorax (<15–20% of hemithorax) can be managed conservatively. Symptomatic patients or patients with a larger pneumothorax need aspiration or drainage.

Chest tube drainage is usually indicated for the following conditions:

- Pneumothorax:
 - In a mechanically ventilated patient
 - Tension pneumothorax after initial decompression by inserting a needle
 - Persistent or recurrent pneumothorax after simple aspiration
- Large or symptomatic pleural effusions
- Other pleural collections:
 - Pus (empyema)
 - Blood (hemothorax)
 - Chyle (chylothorax)
- Postoperative—after thoracotomy or thoracoscopy.

Q. What are the common contraindications to ICD insertion?

Ans.

- Diaphragmatic hernia
- Severe coagulopathy (relative)

- Severe pleural adhesions
- Local site infection.

Q. What is the function of a chest drain?

Ans. Chest drains are inserted to remove air or fluid that collects in the pleural space to allow restoration of negative intrathoracic pressures, and to allow the lung to re-expand.

Other uses of chest drains include:

- The ICD can be used to insufflate air on the ipsilateral side after pneumonectomy in case of mediastinal shift.
- ICD fluid can be tested for amylase levels (to diagnose esophageal rupture or pancreatic lesions) or for chylomicron levels (to diagnose a chyle leak).
- To facilitate pleurodesis.

Q. How does a chest drain function?

Ans. Chest drainage systems work by a combination of the following:

- When the patient breathes out deeply or coughs, the expiratory positive pressure, which is generated helps to push air and fluid out of the chest.
- Positioning the collection chamber of the chest drain below the level of the chest allows fluid drainage by gravity.
- Application of suction to the collection chamber can hasten drainage from the chest; however, suction must be applied cautiously.

Q. What are the components of a chest drainage system?

Ans. Chest drainage systems are made of the following components:

- A chest tube is inserted into the pleural cavity or mediastinal cavity to allow contents (air, fluid) to leave the chest.
- This tube is connected by a length of flexible tubing to the drainage system.
- The drainage system consists of a chamber that collects fluid. There is a one-way seal (fluid level or mechanical valve) that prevents outside air from entering the thoracic cavity during inspiration.

Q. What is the importance of the underwater seal?

Ans. The underwater seal acts as a one-way valve through which air is expelled from the pleural space during expiration and prevented from re-entering during the next inspiration.

Q. Describe the procedure of chest tube insertion.

Ans. Consent should be obtained and all drains should be inserted with full aseptic precautions. The most commonly used position is with the patient lying at 45° with their arm raised behind the head to expose the axillary area.

Thoracic ultrasound guidance is strongly recommended for all pleural procedures for pleural fluid. Small drains should be used first line for pneumothorax, free flowing pleural effusions and pleural infection.

The preferred site for insertion of the needle for pleural aspiration should be the triangle of safety bordered by the anterior border of latissimus dorsi, the lateral border of the pectoralis major, a line superior to the horizontal level of the nipple and an apex below the axilla.

Antibiotic prophylaxis is not recommended for nontrauma patients requiring a chest drain.

Lidocaine 1% should be infiltrated prior to the procedure paying particular attention to the skin, periosteum, and the pleura.

Drains should never be inserted using substantial force. The dilator should not be inserted further than 1 cm beyond the depth from the skin to pleural space. Blunt dissection should be employed in cases of trauma or insertion of large-bore drains.

The procedure should be stopped when no more fluid or air can be aspirated, the patient develops symptoms of cough or chest discomfort or 1.5 L has been withdrawn.

A chest drain may be withdrawn to correct a malposition, but should never be pushed further in due to the risk of infection.

Q. Describe periprocedure analgesia.

Ans.

- Local anesthetic infiltration (2% lignocaine).
- May require postprocedure oral paracetamol, non-steroidal anti-inflammatory drugs (NSAIDs).

- If pain continues after oral analgesics may consider local site infiltration or intercostal block.

Q. What are the available chest tube sizes?

Ans. The size of a chest tube refers to its outer diameter and is given in "French" (Fr), 1 Fr corresponding to one-third millimeter. Chest tube sizes usually range between 8 and 36 Fr. Small-bore chest tubes are defined as 14 Fr or less and large-bore chest tube as >14 Fr. The British Thoracic Society guidelines suggest that in pneumothoraces and effusions of a malignant or an infectious nature (including empyema) small-bore chest tubes are usually adequate, although there is a lack of randomized trial data.

Q. Describe the single-bottle drainage system.

Ans. The drainage tube is submerged to a depth of 1–2 cm in a collection chamber of approximately 20 cm diameter. When the patient inspires, water is drawn up the tube to the height equal to the negative intrathoracic pressure (usually up to 20 cm of H₂O). Therefore, the collection chamber must be kept far enough below the patient to prevent water from being sucked up into the chest. Usually a height of 100 cm is sufficient, to allow for negative pressures as high as 80 cm H₂O. The portion of the tubing that is underwater offers resistance to expiration; this should be no > 2–3 cm. The end of the tube in the underwater seal bottle must remain covered with water at all times. Collected air escapes through a side vent in the chamber (**Fig. 1**).

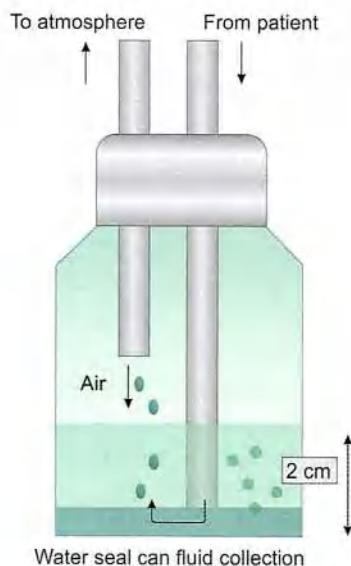


Fig. 1: Single-bottle drainage system.

Q. What is the disadvantage of this single-bottle drainage system?

Ans. As fluid drains from the chest, the level of fluid in the underwater seal starts rising. This increases the resistance against which the patient has to breathe and can obstruct further outflow of fluid from the chest.

Q. Describe the two-bottle drainage system.

Ans. To overcome the disadvantage of the single-bottle system, a second bottle is introduced between the chest tube and the underwater seal. This trap bottle collects the fluid draining out of the chest, while the air passes on to the second bottle and is vented to the atmosphere. This keeps the underwater seal at a constant level (**Fig. 2**).

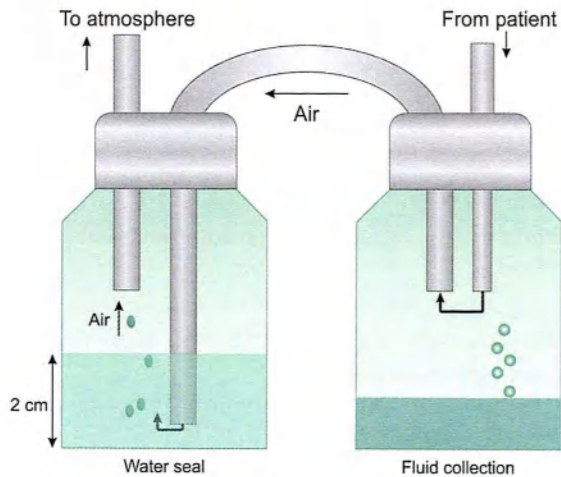


Fig. 2: Two-bottle drainage system.

Q. Describe the three-bottle drainage system.

Ans. When a negative pressure is desired, a third bottle is added between the underwater seal and the suction device. It has an inlet, connected to the vent of the underwater seal chamber of the two-bottle system, an outlet connected to the suction device, and a control tube, which is open to the atmosphere on one side and submerged 20 cm underwater on the other (**Fig. 3**).

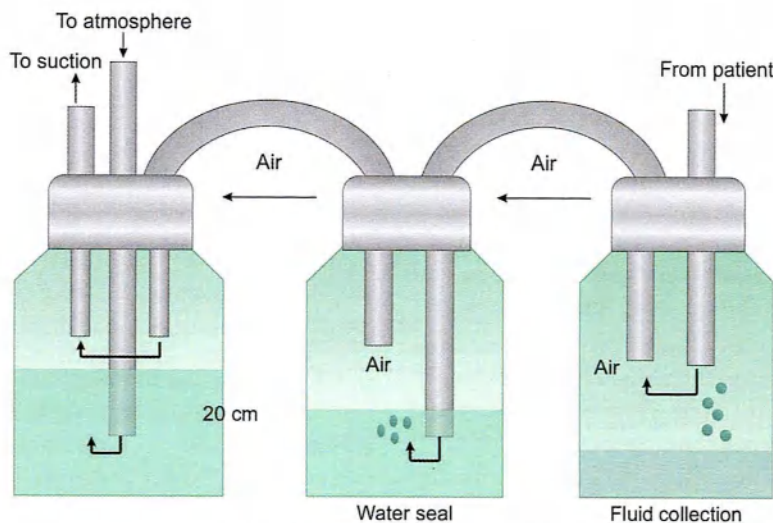


Fig. 3: Three-bottle drainage system.

When suction is applied, air is drawn down the atmospheric vent in this bottle, equal to the pressure inside the bottle that is decreased by the vacuum. This low pressure suction is transmitted to the underwater seal bottle and then into the pleural cavity, thus aiding evacuation of contents and allowing quicker re-expansion of the underlying lung. The maximum force of suction is determined by the depth of the atmospheric vent underwater in the suction regulation bottle. For example, to obtain a suction of 20 cm of H_2O , the tip of the tube should be 20 cm below the surface of the fluid. Turning up the suction does not increase negative pressure within the pleural drainage system.

The three-bottle system provides a fairly stable water-seal level, allows for accurate documentation of the drainage and also controlled suction. Unfortunately, it is bulky and does not allow for easy transport or ambulation of the patient.

Q. How much suction can be applied to the ICD bottle? When should suction be avoided?

Ans. The negative pressure generated depends on the depth of the tube below the water level, and not on the applied suction pressure. Generally pressures between 20 and 40 cm H_2O are achieved.

These bottles can also be used without the application of suction to the third bottle (gravity drainage)—suction is indicated in cases when lung expansion is incomplete, e.g. parenchymal air leaks.

Suction is contraindicated in cases of major air leaks (e.g., bronchopleural fistula) or in massive pleural effusions where controlled and phased drainage is needed to avoid re-expansion pulmonary edema. Suction should also be avoided in post-pneumonectomy patients, in whom a mediastinal shift may be precipitated.

Q: What is the four-chamber pleural drain system?

Ans. In the four-chamber system, an additional underwater seal, vented to the atmosphere is added to the first bottle. This acts as a safety valve in case of suction failure in the three bottle system (please refer to <https://derangedphysiology.com/main/required-reading/equipment-and-procedures/Chapter%20262/underwater-seal-chest-drain-system>).

Q. What is the multifunction chest drainage system?

Ans. The two- and three-bottle drainage systems are bulky and can result in accidental disconnections. Commercially available multifunction drainage systems incorporate all the components of the three-bottle system into a single unit. Examples of multifunction chest drain systems include Pleur-evac (Fig. 4) and Redivac systems.

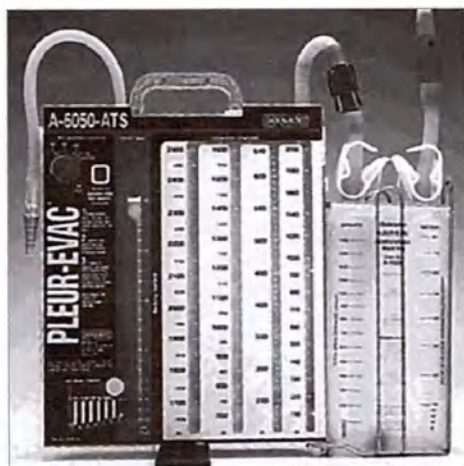


Fig. 4: Multifunction chest drainage system.

Q. What is indwelling pleural catheter?

Ans. This is a drain which allows ambulation in patients with recurrent and symptomatic malignant effusions or refractory effusions due to congestive heart failure. The PleurX catheter is essentially a small-bore chest tube that is designed to remain in place for prolonged, intermittent drainage of the pleural space through a unidirectional flutter valve (i.e., Heimlich valve). The catheters can remain in place for a period of 1–15 months.

Q. Describe the management of the chest tube and precautions that need to be taken during anesthesia.

Ans.

- A bubbling chest tube should never be clamped. Clamping a chest drain in the presence of a continuing air leak may lead to tension pneumothorax.
- Drainage of a large pleural effusion should be controlled to prevent the potential complication of re-expansion pulmonary edema. No more than about 1.5 L should

be drained at one time, or drainage should be slowed to about 500 mL/h.

- The drainage bottle should always be placed below the level of the chest to prevent fluid from re-entering into the thoracic cavity.
- In the presence of a large air leak, loss of tidal volume should be anticipated. These patients should be maintained on spontaneous breathing if possible, until lung isolation can be achieved.

Q. How is air leak assessed with an ICD?

Ans. The presence of air leak should be assessed daily to determine whether the tube could be removed.

Qualitatively, air leaks are classified as follows with increasing severity:

- Forced expiratory—occurs only with coughing or Valsalva
- Expiratory—alveolar-pleural fistulas
- Inspiratory—ventilated patient, bronchopleural fistula, alveolar-pleural fistula
- Continuous—ventilated patient, true bronchopleural fistula.

Q. Three days after insertion of the chest tube, it is found that the fluid column is not moving. What are the possible causes?

Ans. Absence of fluid oscillations can occur when:

- The tubing is clamped or kinked.
- The patient is lying on the tubing.
- There is a dependent, fluid-filled loop in the tubing.
- Lung tissue or adhesions are blocking the catheter eyelets inside the pleural cavity.
- Blockage of the tube due to a blood clot.
- No more air is leaking into the pleural space and the lung has re-expanded completely.

Q. When can an ICD be removed?

Ans. An ICD can be removed when:

- Drainage diminishes to little or nothing.
- Air leak has stopped.
- Fluctuations in the water seal chamber stops.
- The patient is breathing normally without any signs of respiratory distress.
- Breath sounds are equal and at baseline for the patient.
- Chest X-ray shows the lung is re-expanded completely and there is no residual air or fluid in the pleural space.

Q. How is the ICD removed?

Ans. When the tube is ready to be removed, the patient should be asked to perform a Valsalva maneuver (to increase the pleural pressure and prevent air entering the pleural cavity) and the tube is withdrawn quickly. The previously placed suture is then tied to close the hole.

Q. What are the complications of chest drains?

Ans.

- Hemorrhage due to vascular injury while inserting
- Blocked tube due to poor positioning
- Cardiac dysrhythmia
- Infections
- Re-expansion pulmonary edema.

Q. What is re-expansion pulmonary edema?

Ans. Re-expansion pulmonary edema usually occurs unilaterally after rapid re-expansion of a collapsed lung in patients with a pneumothorax. Patients developing re-expansion pulmonary edema typically present soon after chest tube placement with cough, dyspnea, and hypoxemia, but the clinical presentation can be delayed for up to 24–48 hours. Treatment is supportive and the disease is usually

self-limited. It may rarely follow evacuation of large volumes of pleural fluid (>1–1.5 L) or removal of an obstructing endobronchial tumor. The incidence of re-expansion pulmonary edema appears to be related to the rapidity of lung re-expansion and to the severity and duration of lung collapse.

■ SUGGESTED READING

1. Laws D, Neville E, Duffy J; Pleural Diseases Group, Standards of Care Committee, British Thoracic Society. BTS guidelines for the insertion of a chest drain. *Thorax*. 2003;58 (Suppl 2):ii53-9.
2. Tube thoracostomy management. [online] Available from: <http://emedicine.medscape.com/> [Last accessed on February, 2020].
3. Underwater chest drain. [online] Available from: <http://www.frca.co.uk/> [Last accessed on February, 2020].

Hypertensive Disorders in Pregnancy

Sumitra Ganesh Bakshi, Reshma Ambulkar, Shilpushp Jagannath Bhosale

A 24-year-old primigravida, diagnosed with pregnancy-induced hypertension (PIH) on medication, was admitted at 36 weeks for elective section. She has high blood pressure and headache. Discuss perioperative management.

Q. How are the hypertensive disorders related to pregnancy classified?

Ans. Hypertensive disorders of pregnancy can be classified as follows:

- Gestational hypertension [formerly pregnancy-induced hypertension (PIH) or transient hypertension]
- Pre-eclampsia and eclampsia
- Pre-eclampsia superimposed on chronic hypertension
- Chronic hypertension.

Gestational Hypertension

It is said to be present when blood pressure (BP) is $>140/90$ mm Hg for the first time during pregnancy after 20 weeks but no proteinuria. This is transient hypertension and BP becomes normal by 12 weeks postpartum.

Pre-eclampsia or pre-eclampsia toxemia: Pre-eclampsia or pre-eclampsia toxemia is defined as a hypertensive syndrome that occurs in pregnant women after 20 weeks' gestation, consisting of new onset and persistent hypertension (defined as a systolic pressure ≥ 140 mm Hg and/or diastolic BP ≥ 90 mm Hg taken on at least two occasions >4 hours apart with any of the following):

- Urine analysis:
 - Proteinuria [>300 mg/24 hours, or persistent 30 mg/dL (1+ on dipstick) in two random urine samples, collected at least 4 hours apart]
 - Urine albumin: Creatinine ratio can be used as an alternative to urine protein:creatinine ratio to diagnose pre-eclampsia (>8 mg/mmol is diagnostic).
- Evidence of systemic involvement such as renal insufficiency (serum creatinine > 1.1 mg/dL), liver involvement (elevated transaminases and/or right

upper quadrant pain), neurological complications, and hematological complications

- Fetal growth restriction (FGR).

Severe pre-eclampsia is pre-eclampsia with any of the following:

- BP $> 160/110$ mm Hg
- Proteinuria 2.0 g/24 hours or $>2+$ on dipstick
- Serum creatinine > 1.2 mg/dL unless known to be previously elevated
- Platelets $< 100,000/\text{mm}^3$ (sign of worsening pre-eclampsia caused by platelet activation and aggregation and microangiopathic hemolysis induced by vasospasm)
- Microangiopathic hemolysis
- Elevated serum glutamic oxaloacetic transaminase (SGOT)/serum glutamic pyruvic transaminase (SGPT) levels
- Persistent headache and/or visual disturbances
- Persistent epigastric pain (results from hepatocellular necrosis, ischemia, and edema leading to stretch in Glisson's capsule).

Severity of pre-eclampsia: The differentiation between mild and severe pre-eclampsia can be misleading because mild disease may progress rapidly to severe disease (**Table 1**).

TABLE 1: Differences between mild and severe pre-eclampsia.

	Mild pre-eclampsia	Severe pre-eclampsia
Diastolic BP	<100 mm Hg	110 mm Hg or higher
Proteinuria	Trace or 1+	Persistent 2+ or more
Headache	Absent	Present
Visual disturbances	Absent	Present
Upper abdominal pain	Absent	Present

Contd...

Contd...

	Mild pre-eclampsia	Severe pre-eclampsia
Oliguria	Absent	Present
Convulsion	Absent	Present (eclampsia)
Fetal growth restriction	Absent	Obvious
Pulmonary edema	Absent	Present
Serum creatinine	Normal	Elevated
Thrombocytopenia	Absent	Present
Liver enzyme elevation	Minimal	Marked

Chronic hypertension: BP > 140/90 mm Hg before pregnancy or diagnosed before 20 weeks' gestation or hypertension first diagnosed after 20 weeks' gestation and persistent after 12 weeks postpartum.

Superimposed Pre-eclampsia (on Chronic Hypertension)

All chronic hypertensive disorders regardless of their cause predispose to development of superimposed pre-eclampsia or eclampsia. Pre-eclampsia is accompanied by proteinuria.

Eclampsia

Eclampsia is the occurrence of seizures in a woman with pre-eclampsia when the seizures cannot be attributed to other causes. The seizures are grand mal and may appear before, during, or after labor. It may develop >48 hours after delivery and may be encountered up to 10 days postpartum.

Q. Discuss the pathogenesis of pre-eclampsia.

Ans. Risk factors: Increased incidence is found in primigravida, multiple pregnancies, history of chronic hypertension, maternal age > 35 years, obesity [body mass index (BMI) > 30]. Decreased incidence is found in women who smoke and in patients with placenta previa.

Theories for development of pre-eclampsia: Vasospasm is basic to the pathophysiology of pre-eclampsia and eclampsia.

Proposed failure of the normal invasion of trophoblast cells leads to maladaptation of maternal spiral arterioles. The maladaptation of these vessels can interfere with normal villous development leading to placental insufficiency and subsequently hypoxia leading to FGR, and the release of both vasoconstrictors and vasodilators leading to increased perfusion of vascular beds with endothelial damage and hypertension.

Vascular constriction causes resistance to blood flow and accounts for the development of arterial hypertension. It is likely that vasospasm itself causes damaging effect on vessels. The vascular changes together with local hypoxia of the surrounding tissues presumably lead to hemorrhage, necrosis, and other end-organ disturbances. Some theories put forward include:

- **Increased pressor responses:** Women with PIH have been found to have increased vascular sensitivity to pressors and increased sensitivity to angiotensin II preceding the onset of PIH.
- **Prostaglandins:** In PIH, there is decreased prostacyclin production and increased thromboxane A₂, resulting in vasoconstriction and sensitivity to infused angiotensin II.
- **Nitric oxide**, also known as endothelium-derived relaxing factor (EDRF), is a potent vasodilator. Decreased levels are found in PIH patients.
- **Vascular endothelial growth factor (VEGF):** It is important in vasculogenesis and control of microvascular permeability. VEGF has been reported to be increased in serum from women with pre-eclampsia. Increased level in PIH may represent compensatory mechanism to restore uteroplacental blood flow toward normal.
- **Genetic predisposition:** The tendency for pre-eclampsia-eclampsia is inherited.
- **Immunological factors:** PIH is probably an immune response to antigenic sites on the placenta. This may arise where effective immunization by a previous pregnancy is lacking, as in first pregnancies or where the number of antigenic sites provided by the placenta is unusually great (multiple pregnancies).
- **Inflammatory factors:** Pre-eclampsia is considered a disease due to an extreme state of activated leukocytes in the maternal circulation.
- **Endothelial cell activation:** Pre-eclampsia is an immunologically mediated deficiency in trophoblastic invasion of spiral arteries leading to fetoplacental hypoperfusion. These changes in turn provoke activation of the vascular endothelium with the clinical syndrome of pre-eclampsia.

Q. Can pre-eclampsia be predicted and prevented?

Ans. Early predictors of PIH: History of previous pre-eclampsia or another strong clinical marker of increased pre-eclampsia risk, especially multiple pregnancy, antiphospholipid antibody syndrome, significant proteinuria at the first antenatal visit, or a pre-existing condition of hypertension, diabetes mellitus, and renal disease.

A variety of biochemical and biophysical markers have been proposed for the purpose of predicting the development of pre-eclampsia later in pregnancy. All the strategies have low sensitivity for the prediction of pre-eclampsia. Some of the tests are as follows:

- **Rollover test:** The patient is asked to assume supine position after lying laterally recumbent. A positive test is an elevation of 20 mm Hg or more in systolic BP. This test has a very good correlation with the angiotensin sensitivity test.
- **Elevated uric acid levels** > 5.9 mg/dL.

- **Calcium metabolism:** Hypocalciuria seen with pre-eclampsia.
- **Angiotensin II infusion test:** It is difficult to perform and hence not used in routine clinical practice.
- **Decreased urinary kallikrein excretion:** It might precede the development of pre-eclampsia.
- **Increased cellular plasma fibronectin:** These generally precede the development of pre-eclampsia.
- **Coagulation activation:** Thrombocytopenia and abnormal platelet aggregation appear to be an integral feature of pre-eclampsia. Excessive platelet activation has been linked to maternal vasoconstriction, endothelial cell injury, placental infarction (atherosis and FGR), and transient renal dysfunction. Thromboxane A2 is released promoting vasospasm, further platelet aggregation, and endothelial cell injury.
- **Immunological factors:** Levels of tumor necrosis factor- α (TNF- α), growth factors, and interleukins increased.
- **Placental peptides:** Increased corticotropin-releasing hormone, activin A, and inhibin A.
- **Doppler velocimetry studies of the uterine arteries:** It is an unreliable screening test. Sensitivity is 78% with a positive predictive value (PPV) of 28%. However, Doppler ultrasound velocimetry of the uteroplacental circulation cannot be recommended routinely at present for women at low or increased risk of pre-eclampsia until such screening has been shown to improve the pregnancy outcome.

Risk factors for pre-eclampsia: As per NICE (National Institute for Health and Care Excellence) guidelines 2010, the following have been described to be risk factors for pre-eclampsia:

- **High-risk factors (any one):**
 - Hypertension during last pregnancy
 - Chronic renal disease
 - Autoimmune disorders such as systemic lupus erythematosus (SLE) or antiphospholipid syndrome
 - Diabetes
 - Chronic hypertension.
- **Moderate-risk factors (more than one):**
 - First pregnancy
 - Assisted reproduction
 - Age 40 years or more
 - Pregnancy interval of >10 years
 - BMI of 35 kg/m² or more at the first visit
 - Family history of pre-eclampsia
 - Multiple pregnancies
 - Vitamin D deficiency, low dietary calcium.

Preventive measures: Aspirin should be started at 75 mg daily from 12 weeks of gestation till delivery for patients with

any one high-risk factor or more than one moderate-risk factors. Nitric oxide donors, low molecular weight heparin (LMWH), diuretics, and progesterone have no role to play and there are no recommendations for their use. Nutritional supplementation in the form of fish oil, garlic, antioxidants, vitamins C and E, folic acid, and salt restriction have not been shown to prevent PIH. There is inconclusive evidence regarding L-arginine supplementation in prevention of PIH. Calcium supplementation in high doses (>1 g daily) during pregnancy may reduce pre-eclampsia in women with low dietary calcium intake. However, routine calcium supplementation can lead to development of HELLP (hemolysis, elevated liver enzymes, low platelet count) syndrome. Adequate rest in the third trimester and reduction of workload or stress are advisable. A controlled weight loss and exercise during pregnancy should be encouraged.

Measures for prevention of other pregnancy complications are prostaglandin precursors, magnesium supplementation, and heparin to prevent venous thromboembolic disease.

Other recommendations are abstention from alcohol, periconceptual use of a folate-containing multivitamin, and smoking cessation.

Q. What are the pathological alterations that occur due to pre-eclampsia?

Ans.

- **Cardiovascular changes** are basically related to an increase in cardiac afterload due to hypertension.
- **Hematological changes:** Thrombocytopenia (defined as platelet count < 1,00,000/mm³) is likely due to platelet activation and consumption and reduced platelet production. The other changes include micro-angiopathic hemolysis (due to intense vasospasm), decreased clotting factors, increased erythrocyte destruction, increased fibrin degradation products, and deficiency of antithrombin III.
- **Endocrine changes:** Increase in deoxycorticosterone in the third trimester due to conversion from plasma progesterone causes retention of sodium. Sodium retention leads to inhibition of juxtaglomerular apparatus resulting in decreased plasma levels of renin, angiotensin II, and aldosterone as compared with normotensive pregnancy.
- **Fluid and electrolyte changes:** In pre-eclampsia there is decreased plasma oncotic pressure, which leads to increased extracellular fluid with an associated decrease in the intravascular volume.
- **Kidney:** Oliguria is a result of reduced renal perfusion and glomerular filtration. Plasma uric acid and serum creatinine are elevated. There is proteinuria > 300 mg/24 hours or >1+ on a urinary dipstick test. Acute renal

failure (ARF) and renal cortical necrosis may develop in severe cases.

- **Liver:** Pre-eclampsia is often associated with increased liver enzymes. Epigastric and right hypochondriac pain when present is due to periportal hemorrhagic necrosis, which causes hepatic rupture or subcapsular hematoma.
- **HELLP syndrome:** Triad of hemolysis, elevated liver enzymes, and low platelets.
- **Nervous system:** Cerebral edema, hyperemia, focal thrombosis, and hemorrhage may be present in severe cases. Retinal artery vasospasm may cause visual disturbances. Increased cerebral perfusion pressure may cause severe headaches.
- **Uterus and placenta:** Incomplete trophoblastic invasion of spiral arteries causes reduction in the diameter of vessels as compared to normal. In addition, vasospasm decreases placental perfusion leading to intrauterine growth retardation (IUGR).

Q. Describe neonatal complications related to pre-eclampsia.

Ans.

Short-term neonatal complications: Oligohydramnios, FGR, intrauterine fetal death (IUFD), preterm birth, low Apgar score, newborn intensive care unit (NICU) stay.

Long-term neonatal complications: Cerebral palsy, low intelligence quotient (IQ), hearing loss, visual impairment, insulin resistance and diabetes mellitus, coronary artery disease, and hypertension.

Q. Describe medical management of PIH.

Ans.

Medical Management of Pre-eclampsia

- Early prenatal detection and prompt institution of therapy and corrective measures. The timing of prenatal examinations needs to be scheduled at intervals of 4 weeks until 28 weeks and then every 2 weeks until 36 weeks and weekly thereafter.
- Hospitalization is considered for women with new-onset hypertension, which is persistent or worsening or development of proteinuria. A systematic evaluation should include:
 - Detailed examination followed by daily scrutiny of headache, visual disturbance, epigastric pain, and rapid weight gain
 - Weight on admission and every day thereafter
 - Analysis for proteinuria on admission and at least every 2 days thereafter
 - 4-hourly BP monitoring in a sitting position

- Measurement of serum creatinine, complete blood count (CBC), liver enzymes, platelets—frequency determined by the severity of hypertension
- Frequent fetal monitoring clinically or by ultrasonography (USG).
- Ample but not excessive proteins and calories and no salt or fluid restriction in the diet.

Antihypertensive Drug Therapy

Hypertensive women who take acetylcholinesterase (AChE) inhibitors, angiotensin II receptor blockers (ARBs), and chlorothiazides to stop these drugs and change over to other drugs before they become pregnant as these drugs can cause congenital malformations. BP should be controlled to <160/110 mm Hg to prevent maternal morbidity, particularly from intracranial hemorrhage, encephalopathy, myocardial ischemia, and failure. Oral antihypertensive includes the following drugs:

- **β -blockers:** Labetalol (200–2400 mg/day). It is a combination of α - and β -blockers. It is preferred to a pure β -blocker. Important to note: Prolonged β -blockers may reduce fetal growth.
- **Methyldopa:** It is an α -methyl analog of dihydroxyphenylalanine (DOPA), the precursor of dopamine (DA) and noradrenaline (NA). The α -methyl-NA is a selective α_2 agonist formed in the brain from methyldopa and acts on central α_2 receptors to decrease efferent sympathetic activity. Antihypertensive effect develops over 4–6 hours and lasts for 12–24 hours. Dose: 0.25–0.5 g BD–QID oral (maximum 3 g/day).
- **Nifedipine:** It is a calcium channel blocker, which lowers BP by decreasing peripheral vascular resistance without compromising cardiac output. The usual dose is 10–20 mg every 4–6 hourly.

The target of BP control in uncomplicated chronic hypertension is to keep BP < 150/100 mm Hg. Do not lower diastolic BP < 80 mm Hg. In pregnant women with target-organ damage secondary to chronic hypertension, the aim is to keep BP < 140/90 mm Hg.

BP should be lowered to <160 mm Hg systolic and <110 mm Hg diastolic. The initial antihypertensive therapy in the hospital setting should be with nifedipine short-acting capsules, parenteral hydralazine, or parenteral labetalol. Alternative antihypertensive medications include nitroglycerin infusion, oral methyldopa, oral labetalol, oral clonidine, or postpartum, oral captopril. Refractory hypertension may be treated with sodium nitroprusside. Nifedipine and magnesium sulfate (MgSO_4) can be used simultaneously. MgSO_4 is not recommended solely as an antihypertensive agent.

Continuous fetal heart rate (HR) monitoring is advised until BP is stable.

Q. What are the complications associated with PIH?

Ans.

Maternal complications:

- Intracranial hemorrhage (leading cause of maternal death)
- Disseminated intravascular coagulation (DIC)
- Pulmonary edema
- Congestive heart failure (CHF)
- Placental abruption
- Postpartum hemorrhage (PPH)
- ARF
- Liver rupture
- Cerebrovascular accident
- Septic shock.

Fetal complications:

- Prematurity with respiratory distress
- IUGR
- Oligohydramnios
- Intracranial hemorrhage
- Small forage
- Meconium aspiration.

Q. Discuss the consensus bundle adopted in the west to reduce complications associated with eclampsia.

Ans. The National Partnership for Maternal Safety under the guidance of the council on Patient Safety in Women's Health Care has developed a new safety bundle to reduce the complications associated with eclampsia. The bundle includes a set of evidence-based guidelines which can be easily adapted:

- Early recognition by instituting protocols which measure BP and urine protein in all pregnant and postpartum women
- Educating parturients on early signs and symptoms of pre-eclampsia and hypertension
- To have checklist and protocols to ensure adequate and right management of severe pre-eclampsia
- Obstetric anesthesiologist should be involved in BP management and communicating with the obstetrician about the need of invasive lines and intravenous (IV) infusion for BP control
- $MgSO_4$ infusion should not be discontinued during cesarean sections (CS). Discontinuation of infusions can lead to suboptimal levels in the postoperative period and place women at risk for seizures.

Q. What drugs are used for IV control of BP?

Ans. IV control of BP must be done under strict monitoring and careful titration.

Drugs used

Labetalol: Dosing regime—50 mg over 1 minute for rapid control, followed by 20 mg/h, which can be increased by 20 mg every 20 minutes to a maximum of 160 mg/h.

Caution: Same as oral labetalol.

Hydralazine: 5–10 mg by slow IV injection for rapid control, or 200–300 µg/min for rapid control, and then 50–150 µg/min for maintenance control.

Caution: Acute porphyria, cor pulmonale, idiopathic SLE, severe tachycardia.

Patients may need fluid bolus along with titration of antihypertensives.

Q. What conditions mandate immediate delivery?

Ans.

- Severe hypertension that persists after 24–48 hours of treatment
- Progressive thrombocytopenia
- Liver dysfunction
- Progressive renal dysfunction
- Premonitory signs of eclampsia
- Evidence of fetal jeopardy
- Persistent headache or other neurologic sequelae of pre-eclampsia.

Q. Describe management of uncomplicated pre-eclampsia.

Ans. Management of pre-eclampsia depends upon:

- Severity of pre-eclampsia
- Duration of gestation
- Condition of cervix.

When the fetus is known or suspected to be preterm, the tendency is to temporize in the hope that a few more weeks in utero will reduce the risk of neonatal death or serious morbidity. Assessment of fetal well-being is done using nonstress test and biophysical profile. Measurement of the lecithin-sphingomyelin ratio in the amniotic fluid may provide evidence of lung maturity. Glucocorticoids may be tried for fetal lung maturity before 34 weeks of gestation. If birth is likely within 7 days, give two doses of betamethasone 12 mg intramuscularly 24 hours apart in women between 24 and 34 weeks. For a woman near term, if pre-eclampsia is mild but cervix is firm and closed, the pregnancy is allowed to continue under close observation until the cervix is more suitable for induction.

For a woman near term with a soft, partially effaced cervix, and even with milder degrees of pre-eclampsia, labor is induced by carefully monitored oxytocin induction.

In the past, both spinal and epidural were avoided in women with pre-eclampsia and eclampsia due to concerns of severe hypotension induced by sympathetic blockade and dangers from pressor agents or large volumes of IV fluids used to treat hypotension. However, with improvement in the techniques, and increased usage of epidural analgesia (EA), many clinicians feel that EA should be the preferred method. The advantages of EA are as follows:

- Good pain relief
- Attenuation of the exaggerated hypertensive response to the pain of pre-eclamptic women
- Reduced circulating levels of catecholamines and stress-related hormones
- Improved intervillous blood flow in pre-eclamptic women.

Pre-eclamptic women are at increased risk of cesarean delivery compared with normal parturients, and an early administration of EA for labor facilitates the subsequent administration of epidural anesthesia for emergent cesarean delivery.

Q. What are the adverse effects of labor pain and what various techniques are available for pain relief during labor?

Ans.

Adverse Effects of Painful Labor

Painful labor causes maternal hyperventilation. The resulting alkalosis shifts the oxyhemoglobin dissociation curve to the left in the mother. This results in increased binding of maternal hemoglobin to oxygen and reduced oxygen delivery to the fetus.

Hyperventilation at the time of uterine contractions is followed by hypoventilation in between the contractions. This can result in fetal hypoxemia.

Catecholamine release in response to labor pain causes reduction in uterine blood flow.

The hypertensive response to sympathetic stimulation is detrimental in the hypertensive mother.

Sympathetic stimulation causes maternal acidosis because of lactic acid production from skeletal muscle activity and free fatty acid activation. Lactic acidosis can cause incoordinated uterine action and prolonged labor.

Techniques of Labor Analgesia

- *Nonpharmacological techniques:*
 - *Psychoprophylactic approach of Lamaze:* This is based on gate control theory of pain relief. It hypothesizes that through stimulus-response conditioning, women can learn to use controlled breathing to reduce the pain in the labor. So, the mother is taught to concentrate and is conditioned to respond to contractions with relaxation using breathing techniques to reduce pain.
 - *Leboyer technique:* In this method the baby is allowed to be born in a quiet, dimly lit room, with minimal noise to reduce the trauma and stress for the newborn:
 - Transcutaneous electrical nerve stimulation (TENS)
 - Aromatherapy
 - Hypnosis

- Acupuncture
- Reflexology.

- *Pharmacological techniques:*

- *Entonox:* This is a 50% mixture of nitrous oxide in oxygen. It is delivered to the patient via a demand valve through a low-resistance breathing system from the cylinder after pressure reduction:
 - The advantages are that it has rapid onset and elimination.
 - The disadvantages are that this technique is not adequate for forceps delivery, episiotomy, perineal suturing, or removal of retained placenta.
 - There is also a concern about the pollution of the labor suite, though scavenging devices can be used.
 - Below 6°C the nitrous oxide may remain in the liquid phase allowing the oxygen to be exhausted first after which the cylinder will deliver only 100% nitrous oxide. Guidelines laid down for storage and handling of these cylinders should be followed.
- *Sevoflurane:* Patient-controlled inhalation analgesia. Sevoflurane is a volatile inhalational agent commonly used during general anesthesia (GA). Because of its short onset and offset of action, it appears to be the best-suited inhalational agent for labor analgesia and can be administered as patient-controlled inhalation analgesia. It is used in the concentration of 0.8% with oxygen and needs specialized equipment. Further, there is a concern for environmental pollution and maternal amnesia and loss of protective airway reflexes. Larger studies are needed to assess the incidence of maternal compromise.
- *Opioids:* In the United Kingdom, pethidine is commonly used for labor analgesia and can be prescribed by the midwives. It is less depressant for the baby than morphine. It is used in the dose of 100–150 mg intramuscularly. Pethidine 25 mg IV and nalbuphine 10 mg IV are also used. The side effects are as follows:
 - *Maternal:* Opioids produce nausea, vomiting, decrease in gastric motility, respiratory depression, disorientation, and disinhibition in the mother. Gastric emptying can be a problem if the mother subsequently needs a GA for an emergency surgery.
 - *Neonatal:* The newborn can have respiratory depression, central nervous system (CNS) depression, and impaired temperature regulation. The respiratory depression has to be reversed with naloxone 0.01 mg/kg (20 µg injected

into the umbilical vein). Maximal neonatal depression is seen when the baby delivers 3 hours after the dose of pethidine is given. The neurological depression can be demonstrated up to 3 days after birth. The metabolite of pethidine (norpethidine), which is produced in the fetoplacental unit, may contribute to this delayed action. The elimination half-life of pethidine in the neonate is 18 hours and that of norpethidine is 60 hours. Recently, short-acting opioids such as fentanyl and remifentanyl are being used as continuous IV infusion or as patient-controlled infusion under medical supervision. These techniques are useful for patients in whom regional blocks are contraindicated due to various reasons such as bleeding diathesis, infection, or allergy to local anesthetics.

- **EA:** This is the most effective method of producing pain relief in labor. Maternal satisfaction and neonatal outcome (as assessed by Apgar scores, umbilical cord pH, and need for naloxone) are better in patients receiving EA.
- **Subarachnoid block:** This is not a suitable technique to produce pain relief during labor as the duration of block is too short. Combined spinal epidural (CSE) is preferred as it has the advantages of immediate pain relief and prolongation of action by using the epidural catheter.

Q. How does the EA work? What are the indications and contraindications to EA?

Ans. During the first stage of labor, pain is felt due to uterine contractions and dilatation of the cervix. It is felt in the cutaneous distribution of T10-L1 dermatomes, i.e., lower abdomen, groins, and lower back. During the second stage of labor, stretching, distension, and tearing of fascia, skin, and subcutaneous tissues as well as the pressure of the presenting part on the perineum cause pain (S2-S4).

These pain fibers are blocked using local anesthetics in dilute concentration so as to cause sensory block and not motor blockade.

Indications

Maternal:

- Maternal request
- Expectation of operative delivery (e.g., multiple pregnancy, malpresentation)
- Any condition where the stress response to labor is to be avoided, e.g., hypertensive, diabetic, pre-eclampsia
- Maternal anxiety and pain leading to prolonged labor, incoordinated uterine action

- Conditions in which GA may be life threatening, particularly if rapid regional anesthesia may be difficult to institute (e.g., morbid obesity).

Contraindications:

- Maternal refusal
- Uncorrected hypovolemia
- Local and systemic sepsis
- Bleeding diathesis
- Fetal distress.

Relative contraindications:

- Expectation of significant hemorrhage
- Untreated systemic infections
- Previous back surgery.

Q. Describe techniques of labor analgesia.

Ans. Prerequisites: There should be an adequate number of trained anesthetists, midwives, and obstetricians to care for the mother's safety.

Resuscitation equipment should be available in every labor room.

A large-bore IV line should be secured to preload the patient with fluids and to manage complications.

The patient should be in active labor with regular painful contractions.

Studies have shown that there is no need to wait for cervical dilatation to reach to an arbitrary 4–5 cm and maternal request is a sufficient indication for initiation of labor analgesia.

Informed consent is obtained, and the obstetrician is consulted.

Monitoring includes the following:

- BP every 1–2 minutes for 15 minutes after giving a bolus of local anesthesia.
- Continuous maternal HR during the procedure.
- Fetal HR monitoring.
- Hydration with 500–1000 mL of Ringer's lactate (RL) (coload).
- The woman assumes a lateral decubitus or sitting position.
- The epidural space is identified with a loss of resistance technique.
- The epidural catheter is threaded 3 cm into the epidural space.
- A test dose of 3 mL of 1.5% lidocaine or 0.25% bupivacaine with 1:200,000 epinephrine is injected. There are reservations to use epinephrine in the test dose. Another method for ruling out intravascular placement is using 100 µg of fentanyl epidurally and watch for drowsiness and euphoria, which is seen within 5–10 minutes in case the catheter is accidentally placed in the intravascular space (sensitivity 92.4% and specificity 92%).

- If the test dose is negative, a bolus dose of 0.065–0.125% bupivacaine is injected to achieve a cephalad sensory level.
- After 15–20 minutes, the block is assessed using loss of sensation to cold or pinprick. If no block is evident, the catheter should be reinserted. If the block is asymmetrical, the epidural catheter should be withdrawn 0.5–1.0 cm and an additional 3–5 mL of bupivacaine injected. If the block remains inadequate, the catheter should be reinserted.
- Caval compression should be strictly avoided throughout labor, as the hypotension due to epidural may be aggravated due to aortocaval compression. The patient has to be nursed in sitting or lateral position.
- Maternal BP is recorded every 5–15 minutes. The fetal HR is monitored continuously.
- The level of analgesia and intensity of motor block should be assessed hourly.

It is important to remember that the operator should use the technique that he/she is most familiar with, i.e., position of the patient—sitting/lateral, loss of resistance to saline or air. For a patient in labor, maternal HR variability during acute uterine contraction may lead to misinterpretation; hence, it is important to inject between contractions. The oral intake of modest amounts of clear liquids may be allowed for uncomplicated patients in labor. The volume of liquid ingested is less important than the type of liquid ingested. However, patients with additional risk factors for aspiration (e.g., morbidly obese, diabetic, difficult airway) or patients at increased risk for operative delivery (e.g., nonreassuring fetal HR pattern) may have further restrictions of oral intake determined on a case-by-case basis.

Epidural block to T10 is needed for labor and to level T4 for CS. The American Society of Regional Anesthesia and Pain Medicine recommendations for neuraxial anesthesia in the presence of LMWH should be followed. Needle placement should occur at least 12 hours after the LMWH dose, whereas patients receiving higher doses of LMWH (e.g., enoxaparin 1 mg/kg twice daily) will require longer delays (24 hours). If LMWH is to be given after a spinal or epidural anesthetic, then the first dose of LMWH should be administered not earlier than 24 hours postoperatively. Catheter removal should be delayed for at least 10–12 hours after a dose of LMWH. Again, subsequent dosing should not occur for at least 2 hours after catheter removal.

Q. Discuss the role of ultrasound in EA.

Ans. Ultrasound may be used to locate the epidural space in obese patients and in patients with abnormal spines. It helps in guiding the needle depth and minimizes failure rates.

Q. Discuss the drugs used for EA and monitoring that is employed.

Ans. Bupivacaine is the local anesthetic of choice. It is a long-acting, lipid-soluble weak base. It is variably bound to alpha-1 acid glycoprotein and therefore the fetal transfer is not significant after epidural administration. Bupivacaine can be used as repeated boluses at an interval of 60–90 minutes or as continuous infusion. 0.5% bupivacaine causes a fairly dense block and is not used for labor analgesia. It is used for interventions such as forceps delivery, perineal tear suturing, or CS. 0.065–0.125% bupivacaine has been used to produce labor analgesia. The tone and power of abdominal and pelvic muscles are better preserved when dilute solutions are used. The quality of analgesia can be improved by combining bupivacaine with a small dose of an opioid. Maintenance of the block with a continuous infusion, or patient-controlled epidural analgesia (PCEA) with a background continuous infusion, provides more stable analgesia and the fluctuation in BP is also reduced.

Newer drugs include the following:

- *Ropivacaine:* It is less cardiotoxic and has a wider safety margin. The protein binding is 94%. For ambulatory analgesia, 0.08% ropivacaine is used with 2 µg of fentanyl. However, clinical studies have failed to show any advantage over bupivacaine.
- *Levobupivacaine:* It is an S-enantiomer of bupivacaine; hence, it is less cardiotoxic. More evidence is needed for this drug.
- *PCEA:* The quality of analgesia is similar to that obtained with other forms of epidural administration. PCEA is a form of epidural and, as such, has the same safety requirements. PCEA has not shown to reduce the workload of the anesthetic team but involves additional cost of the PCA pump.
- *Computer-integrated PCA* has been developed where the computer controls the background infusion rates depending on the previous hour's demand boluses.

Monitoring during labor in a patient with epidural:

- *The patient's position* should be sitting, lateral, or supine with a wedge under pelvis to avoid aortocaval compression.
- *Arterial BP* should be monitored every 5 minutes for the first 20 minutes after every bolus. Thereafter, the BP should be monitored every 30 minutes.
- IV infusion should be running satisfactorily.
- Fetal heart sounds (FHS) are to be monitored continuously.
- The mother must never be left unattended.

Q. What do we mean by walking (mobile) or ambulatory epidural?

Ans. This technique basically involves the use of low dose of local anesthetic in combination with a small

dose of opioid. It could be a CSE technique or only epidural technique. Bupivacaine 1–2.5 mg with fentanyl 10–25 µg has a rapid onset and lasts for 90–120 minutes. This is followed by epidural infusion. Morphine 100 µg can be used intrathecally, but it is associated with a higher incidence of nausea and vomiting. The maternal satisfaction is better in this technique because the motor power is preserved and the mother can ambulate during labor. Some studies have claimed that ambulation may help with cervical dilatation and engagement of the presenting part. However, other studies found that walking or sitting did not shorten labor duration from the time of epidural insertion to complete cervical dilatation. Though the power, coordination, and reflexes in the lower limb are preserved, distal proprioception and vibration sense may be affected. Hence, whenever the mother is ambulating, she should be accompanied by a responsible adult.

Q. Enumerate the complications of EA.

Ans.

Mild complications:

- Shivering
- Intrapartum fever
- Mild itching with the use of opioids
- Backache—it is often not attributable to epidural
- Missed segment or unilateral block
- Urinary retention
- Local anesthetic toxicity.

Major complications:

- *Prolonged labor:* It has been shown that the mean duration of labor is longer by an hour in patients who have epidural as compared to those who do not have one. Oxytocin is used twice as frequently in the epidural group than in patients receiving IV analgesia:
- Hypotension
- Dural tap (0–2.6%)
- High block
- Bloody tap
- Neurological damage
- *Assisted delivery:* Increase in the rate of instrumental deliveries (forceps/vacuum) has been shown in patients receiving EA. However, no relationship has been shown between EA and the rates of CS.

Q. How will you treat hypotension that occurs after EA or when anesthesia is instituted?

Ans.

- Administration of oxygen with facemask
- Left uterine displacement (LUD)
- Increased hydration
- Elevation of lower extremities to facilitate venous return
- Administration of small increments of 2–5 mg ephedrine or phenylephrine 50–100 mg IV, if blood pressure falls >20% of baseline.

Q. How would you manage an accidental dural tap?

Ans. Immediate management: Pass the epidural catheter into intrathecal space and label, and use as an intrathecal catheter OR reinsert the epidural one interspace higher. Run epidural as usual but beware of the possibility of intrathecal spread of local anesthetic.

Prophylactic Treatment

Bed rest: Because of the risk of deep vein thrombosis (DVT) and thromboembolism, bed rest should not be routinely encouraged in asymptomatic women.

Epidural infusion of saline will compress the dural sac and alleviate symptoms. After continuous infusion, the incidence of postdural puncture headache (PDPH) is marginally reduced. However, there is no strong evidence to support use of epidural infusions. *Prophylactic epidural blood patch* has a lower success rate and as bacteremia is common after delivery it has to be used cautiously.

Symptomatic treatment of PDPH:

- Simple analgesics should be started round the clock
- Plenty of oral fluids.

Caffeine or theophyllines: These act by reducing intracranial vasodilatation. Both oral and IV caffeine are efficacious. A suitable regimen is 600 mg caffeine/day in divided doses.

Sumatriptan (5HT agonist): This leads to cerebral vasoconstriction and hence provides relief from symptoms. It is given 6 mg subcutaneously. However, it is expensive and can cause coronary artery spasm.

Adrenocorticotrophic hormone (ACTH) acts by increasing concentration of β-endorphin and intravascular volume.

Abdominal binders: Relieve symptoms of PDPH by increasing epidural vein blood flow, which in turn compresses the dural sac.

Blood patch: Epidural blood patch performed around 48 hours postpartum has a 60–90% cure rate at the first attempt.

Mechanism of action: Injected blood compresses the dural sac and raises the intracranial pressure. This produces an almost instantaneous improvement in pain. Later, the injected blood forms a clot over the site of the dural tear and this seals the cerebrospinal fluid (CSF) leak.

Technique of Epidural Blood Patch

The patient's consent must be obtained. Two operators are required. The procedure should be done in aseptic technique both at the epidural site and at the site of bloodletting, usually the antecubital fossa. An epidural should be performed at the same or a lower space as the dural puncture, with the patient in lateral position. Once the space is identified, 20–30 mL of blood is injected slowly through the epidural needle until the patient complains of pain (lancinating pain of dermatomal

origin) or to a maximum of 20–25 mL. To allow the clot to form, maintain bed rest for 2 hours after the procedure. A simultaneous blood sample for culture should be obtained while collecting blood for the blood patch. Postprocedure backaches are common. Other complications of epidural blood patch include neurological deficits, epileptiform fits, and cranial nerve damage.

Q. How will you diagnose and treat bupivacaine toxicity?

Systemic and localized toxic reactions can occur due to accidental intravascular or intrathecal injection or administration of a large dose of bupivacaine.

Local toxicity: Local anesthetic drugs employed rarely produce localized nerve damage.

Systemic toxicity: Systemic reactions to local anesthetics involve primarily the CNS and cardiovascular system (CVS). Bupivacaine is reported to cause rapid and profound cardiovascular depression. The cardiovascular collapse/CNS ratio is lower for bupivacaine compared to lignocaine.

Cardiovascular signs:

- Direct cardiac effects:
 - Myocardial depression, cardiac dysrhythmias, and cardiotoxicity in pregnancy
- Peripheral effects:
 - Vasoconstriction at low doses
 - Vasodilatation at higher doses (hypotension)
- The range of signs and symptoms of cardiovascular toxicity includes the following:
 - Chest pain
 - Shortness of breath
 - Palpitations
 - Light headedness
 - Diaphoresis
 - Hypotension
 - Syncope.

Central nervous system signs:

- Initial signs and symptoms:
 - Light headedness
 - Dizziness
 - Visual and auditory disturbances (difficulty in focusing and tinnitus)
 - Muscular twitching and tremors initially involving muscles of face and distal parts of the extremities
 - Disorientation
 - Drowsiness
- Higher dose symptoms:
 - Often occur after an initial CNS excitation followed by a rapid CNS depression
 - Convulsions.

Allergic manifestations: Allergic manifestations of local anesthetics include rash and urticaria. Anaphylaxis due to

local anesthetics is very rare but should be considered if the patient is wheezing or in respiratory distress following administration.

Management of toxicity:

- Airway management
- Cardiovascular resuscitation
- Suppression of seizure activity.

Administration of lipid emulsion: Bolus 1.5 mL/kg IV over 1 minute. Continuous infusion of 0.25 mL/kg/min (500 mL over 30 minutes). The bolus can be repeated after 5 minutes and infusion doubled if CVS remains collapsed. The infusion should be continued for 30 minutes.

Q. What are the signs of impending eclampsia?

Ans.

- Headache
- Visual disturbance, such as blurring or flashing before the eyes
- Epigastric pain
- Oliguria
- Vomiting
- Sudden swelling of the face, hands, or feet
- Progressive deterioration in blood tests (such as rising creatinine or liver transaminases or falling platelet count)
- Abnormal Doppler findings.

Q. What measures can be taken to prevent seizures?

Ans. It has been proved that MgSO_4 is superior to phenytoin in preventing eclamptic seizures. There is little evidence to support its prophylactic administration to women with mild disease. Unmeasured fetal effects of maternal eclampsia justify attempts to prevent as well as treat eclampsia in women at high risk for convulsions.

The dosage schedule of MgSO_4 for severe pre-eclampsia is the same as for eclampsia.

Use the Collaborative Eclampsia Trial regimen for administration of MgSO_4 , i.e., loading dose of 4–5 g should be given intravenously over 5 minutes, followed by an infusion of 1 g/h maintained for 24 hours.

If the woman has had an eclamptic seizure, the infusion should be continued for 24 hours after the last seizure and should not be discontinued during delivery.

Recurrent seizures should be treated with a further dose of 2–4 g given over 5 minutes. Measure Mg level at 4–6 hours and adjust infusion to maintain levels between 4 and 7 mEq/L. MgSO_4 is discontinued 24 hours after delivery. It is recommended not to use diazepam, phenytoin, or lytic cocktail as an alternative to MgSO_4 in eclampsia.

Q. How would you monitor and manage MgSO_4 toxicity?

Ans. In therapeutic doses, MgSO_4 is associated with muscle weakness. This is as a result of decrease in the

amount of acetylcholine released at motor nerve endings, decreased action of acetylcholine on the motor end plate, and reduction of excitability of the motor fiber membrane. Therefore, it increases the sensitivity of the mother to both the depolarizing and the nondepolarizing muscle relaxants. When a patient is started on MgSO_4 , it is necessary to monitor the following:

- Patellar reflex (disappears when the plasma magnesium level reaches 10 mEq/L; warning impending Mg toxicity)
- Rate and depth of respiration (depressed at level >10 mEq/L, respiratory paralysis and arrest occur >12 mEq/L)
- Urine output (Mg is cleared totally by renal excretion, when there is renal insufficiency; plasma magnesium level needs to be checked periodically and dosage adjusted accordingly) (Table 2).

TABLE 2: Plasma magnesium levels in mEq/L and clinical effects.

Serum magnesium (mEq/L)	Clinical manifestation
1.5–2.0	Normal plasma level
4–8	Therapeutic level
5–10	Electrocardiogram (ECG) changes (wide QRS, >PQ) loss of deep tendon reflexes Sinoatrial and atrioventricular nodal block Respiratory paralysis
12	Cardiac arrest

Other Effects

Uterine effects: In high concentration, MgSO_4 depresses myometrial contractility secondary to inhibition of intracellular free Ca^{++} concentration in the uterine cell (seen at a serum Mg level of 8–10 mEq/L).

Neonatal effects: Magnesium crosses the placenta to achieve equilibrium in fetal serum. MgSO_4 is associated with a minimal risk of intracranial bleed and irregular bone deposits. It can cause a transient reversible change in the fetal HR and should not be considered as a sign of fetal distress. The neonate may be depressed only if there is severe hypermagnesemia at delivery.

Treatment:

- Withhold MgSO_4
- Administer calcium gluconate, 1 g intravenously.

For severe respiratory depression and arrest, prompt tracheal intubation and mechanical ventilation are lifesaving.

Q. How will you treat eclampsia?

Ans. Previous regimens such as lytic cocktail (IV Pethidine 100 mg + Phenergan 50 mg + Largactil 50 mg) are no longer recommended.

Use the Collaborative Eclampsia Trial regimen for administration of MgSO_4 , i.e., a loading dose of 4 g should

be given intravenously over 5 minutes, followed by an infusion of 1 g/h maintained for 24 hours. Recurrent seizures should be treated with a further dose of 2–4 g given over 5 minutes. General care includes protection of airway and support of ventilation, intermittent IV, or oral administration of antihypertensive medications to lower the BP, judicious fluid management, and delivery of the baby.

Q. How will you manage a patient with eclampsia on MgSO_4 with fetal distress who is posted for emergency lower segment cesarean section (LSCS)?

Ans. Aspiration prophylaxis:

- Preinduction administration of nonparticulate antacid such as 0.3 M sodium citrate, 30 mL
- IV administration of histamine receptor blocking agent 40 minutes before induction
- Administration of metoclopramide to decrease gastric volume 30–60 minutes before induction

Preoxygenation and denitrogenation with 100% oxygen over 3 minutes are the preferred technique; however, if not feasible, the patient can be advised to take 8 vital capacity breaths of 100% oxygen in 1 minute.

If BP is high, rapid control of BP can be achieved by:

- Hydralazine (5 mg IV aliquots up to a maximum of 20 mg)
- Labetalol (5–10 mg IV every 10 minutes)
- Oral nifedipine (sublingual nifedipine should be used with caution because of associated rapid changes in placental circulation, which may compromise fetal condition).

In resistant cases, infusion of sodium nitroprusside and glyceryl trinitrate may be needed. But in these cases, continuous arterial pressure monitoring should be instituted.

To prevent sudden hypotension, crystalloid fluid bolus of 500 mL before or at the same time as the first dose of IV hydralazine in the antenatal period should be given.

Consider arterial line placement before induction in patients with severe pre-eclampsia.

Not every patient needs central venous access. It is indicated in patients with persistent oliguria and presence of pulmonary edema for fluid management in antepartum and postpartum period.

Anticipate difficult airway and keep small-sized tubes, gum elastic bougie, laryngeal mask airway (LMA), and difficult airway set ready. Plan for attenuation of response to laryngoscopy.

Crash induction using thiopentone sodium or propofol (if BP is high) and succinylcholine after checking ability to ventilate.

MgSO_4 potentiates the effects of both depolarizing and nondepolarizing muscle relaxants. One may consider the use of nondepolarizing muscle relaxant, atracurium, for

maintenance of neuromuscular block once the patient has demonstrated recovery from succinylcholine or small intermittent doses of succinylcholine can be used. It is preferable to use a peripheral nerve stimulator in the intraoperative period.

Use two-third of the minimum alveolar concentration (MAC) of the inhalation agent to ensure adequate depth of anesthesia (MAC requirement decreases in pregnancy).

Watch for hemodynamic changes during delivery and removal of placenta. Fluid boluses should be given if hypotension develops.

There is a chance of decreased uterine contractility causing increased bleeding in view of MgSO_4 therapy.

Continue MgSO_4 during intraoperative and postoperative periods.

Q. How will you attenuate pressor response during laryngoscopy and endotracheal intubation?

Ans. We can use a wide variety of drugs and techniques to counteract the cardiovascular responses to laryngoscopy and endotracheal intubation. The ideal anesthetic drug and method should have a rapid onset of action, be safe and convenient to prepare and use, and have duration of action to fit the particular situation.

Lignocaine: IV lignocaine occasionally blunts the BP response and almost never limits the HR response during intubation. If administered, lignocaine should be given at a dose of 1.5 mg/kg IV 3 minutes in young healthy patients and 4 minutes in geriatric patients prior to laryngoscopy.

IV induction agents: Propofol may be a better IV induction drug than thiopental, etomidate, methohexital, or ketamine to attenuate the BP effects of laryngoscopy and intubation, especially if the patient is hypertensive at the time of induction. Concomitant use of a low-dose opioid (fentanyl 1.5–3.0 $\mu\text{g/kg}$) in conjunction with an IV induction drug helps to attenuate the hemodynamic response.

Fentanyl: The recommended IV doses for attenuating the BP and HR are 5–6 $\mu\text{g/kg}$ in young healthy patients and 1.5–3.0 $\mu\text{g/kg}$ in elderly patients administered 3–4 minutes prior to laryngoscopy and endotracheal intubation. These doses may produce respiratory depression and delayed awakening in short surgical cases.

Sufentanil: 0.5–1 $\mu\text{g/kg}$ should be given intravenously 2 minutes prior to laryngoscopy and intubation.

Alpha-agonists: Clonidine is the preferred alpha-agonist to administer at a dose of 5 mg/kg (up to a maximum dose of 300 mg) orally 90 minutes prior to anesthesia induction. The drawback is that it has to be administered orally, necessitating adequate preoperative preparation time, a prolonged duration of action, possible postoperative

sedation, and an increased incidence of perioperative bradycardia and hypotension.

Vasodilators: The vasodilators attenuate the BP but not HR effects of laryngoscopy and intubation. A low dose of a vasodilator combined with a short-acting beta-blocker may provide a possible solution to control both BP and HR. Nitroprusside 1–3 $\mu\text{g/kg}$ IV or nitroglycerin 1.5–2.5 $\mu\text{g/kg}$ IV can be started 15 seconds prior to laryngoscopy and intubation.

Magnesium sulfate: MgSO_4 40 mg/kg IV 1 minute prior to laryngoscopy attenuates the BP but not the HR response to laryngoscopy and intubation.

Calcium channel blockers attenuate the BP but not the HR response to laryngoscopy and intubation. Diltiazem 0.2–0.3 mg/kg or verapamil 0.1 mg/kg may be given 1 minute prior to intubation.

Beta-blockers: Labetalol 0.4–0.75 mg/kg IV 2–5 minutes or esmolol 1.5–2.0 mg/kg IV 2 minutes prior to laryngoscopy and intubation are recommended to attenuate the HR and BP effects.

Q. Should ergot alkaloids be given to the mother after delivery of the baby?

Ans. Pre-eclampsia is a relative contraindication to use of ergot alkaloids because of the risk of hypertensive crisis. In pre-eclamptic patients, uterine atony unresponsive to oxytocin may be treated with 15-methylprostaglandin $\text{F}_{2\alpha}$ (misoprostol) given directly in the uterus.

Q. Are there any concerns with respect to fetal resuscitation in these cases?

Ans. Neonatal resuscitation equipment needs to be kept ready.

Prior information to NICU and neonatologist standby is preferred.

The neonate may be hypotonic (if there is severe hypermagnesemia at delivery).

Problems related to preterm baby:

- Respiratory depression due to high dose of opioids given to mother during GA (naloxone needs to be kept ready)
- In the absence of a neonatologist, and in case of a single anesthesiologist, it is the duty of the anesthetist to first attend to the mother and then to the neonate.

Q. Can spinal anesthesia be considered in the above case?

Ans. It is now widely recognized that epidurals provide relatively smooth control of BP, maintain or improve uteroplacental perfusion, optimize fetal outcome, and eliminate the airway and hemodynamic problems associated with GA. Epidural anesthesia is the current technique of choice among most obstetric anesthetists for CS in severe pre-eclampsia. Good communication between the anesthetist and the obstetrician would help to

anticipate CS need early, and the epidural catheter can be inserted and topped up in good time. However, in the case of urgent CS where it is not appropriate to wait for the time required to produce effective epidural blockade, the choice of technique lies between spinal anesthesia and GA.

There has been a concern of bleeding in view of low platelet count in these patients and also an increased incidence of hypotension following spinal anesthesia. However, it is seen that the incidence of hypotension during spinal anesthesia for cesarean delivery is quite less in pre-eclamptic patients and that this is primarily attributable to pre-eclampsia-associated factors. The bleeding risk during epidural anesthesia is greater than that of lumbar puncture.

The most important factor to be considered before regional analgesia is the trend in platelet count.

As per the NICE guidelines (2018):

For women with gestational thrombocytopenia:

- **Platelet count $> 80 \times 10^9/L$:** No change in birth plan is needed. The woman is treated as any other healthy parturient.
- **Platelet count $50\text{--}80 \times 10^9/L$:** Decision to be made based on risk versus benefit evaluation inclusive of a patient's clinical history, other blood parameters [inclusive of international normalized ratio (INR)], her preferences, and anesthetic expertise.
- **Platelet count $< 50 \times 10^9/L$:** Regional analgesia and anesthesia is to be avoided.

Q. What is HELLP syndrome?

Ans. HELLP syndrome is a severe form of pre-eclampsia characterized by:

- **H**—hemolysis (abnormal peripheral blood smear and an increased bilirubin level of 1.2 mg/dL or more)
- **EL**—elevated liver enzymes [SGOT > 70 U/L, lactate dehydrogenase (LDH) > 600 U/L]
- **LP**—low platelets ($< 1,00,000/mm^3$).

It can occur antepartum or postpartum. Delivery represents the only definitive treatment of HELLP syndrome. Patients with HELLP syndrome have a high incidence of serious maternal complications including DIC, placental abruption, need for blood transfusion, pleural effusion, ARF, and wound infection. After delivery of placenta, recovery from HELLP syndrome can be expected to start within 24–48 hours. These patients should be monitored in the intensive care unit (ICU) or high-dependency unit (HDU). Steroids or plasma exchange have no role in the acute management of HELLP syndrome.

The recommendations for platelet transfusion in a patient with HELLP syndrome in labor are as follows:

- For a platelet count of 20×10^9 to $49 \times 10^9/L$ with hemolysis, elevated liver enzymes, and low platelet syndrome, platelet transfusion is recommended prior to cesarean delivery.

- For a platelet count of 20×10^9 to $49 \times 10^9/L$ with hemolysis, elevated liver enzymes, and low platelet syndrome, platelet transfusion should be considered prior to vaginal delivery if there is excessive active bleeding, known platelet dysfunction, rapidly falling platelet count, or coagulopathy.
- For a platelet count of $\geq 50 \times 10^9/L$ with hemolysis, elevated liver enzymes, and low platelet syndrome, platelet transfusion and/or packed red blood cells should be considered prior to either cesarean or vaginal delivery only if there is excessive active bleeding, known platelet dysfunction, a rapidly falling platelet count, or coagulopathy.

Q. What are the concerns in the postpartum period?

Ans. Provide adequate analgesia using titrated doses of opioids such as tramadol. Nonsteroidal anti-inflammatory drugs (NSAIDs) should be avoided in view of renal dysfunction and low platelets. Paracetamol should be used carefully in patients with deranged liver enzymes. Monitor urine output. $MgSO_4$ should be continued for at least 24 hours postpartum. Maintain hemodynamic control with antihypertensives.

All antihypertensive agents have the potential to transfer into breast milk; therefore, the BP of babies should be monitored, especially those born preterm, for the first few weeks.

Nifedipine/amlodipine or enalapril can be considered for treating hypertension in women during the postnatal period with appropriate monitoring of maternal renal function and serum potassium levels. If BP is not controlled even with a combination of amlodipine and enalapril, atenolol can be added. No diuretic or ARB should be used to treat hypertension postpartum.

Q. What points would you remember while giving cardiopulmonary cerebral resuscitation (CPCR) to a pregnant woman?

- **Primary ABCD survey:** A minimum of four basic life support (BLS) responders should be present
- Circulation
- The patient should be placed on a hard surface and always in supine position
- Manual LUD should be performed on all BLS scenarios in pregnant women in whom the uterus can be palpated, to relieve aortocaval compression during resuscitation
- Defibrillation
- No modifications in dose or pad position. Anterolateral defibrillator pad placement is recommended, with the lateral pad placed beneath the breast tissue
- Defibrillation shocks transfer no significant current to the fetus
- Remove any fetal or uterine monitors before shock delivery.

Airway and breathing:

- Ensure bag mask ventilation with 100% oxygen.
- Two-handed bag mask ventilation is more effective and should be used as soon as the second provider is available.
- If there is no chest rise with bag mask ventilation, one must reopen airway and improve seal; if necessary, use an oral airway.
- Cricoid pressure may not be effective at preventing aspiration, and chest compressions, oxygenations, and relief of aortocaval compression are of higher priority than techniques to limit risk of regurgitation.

Secondary ABCD survey:

- Airway edema and swelling may reduce the diameter of the trachea. An endotracheal tube of diameter 6–7 mm should be used.
- A provider experienced in intubation should insert the tracheal tube. Limit the attempts to two attempts. Supraglottic airway placement is the preferred rescue strategy for failed intubation.
- Interruptions in chest compressions should be minimized during advancement in airway management. Follow standard advanced cardiac life support (ACLS) recommendations for administration of all resuscitation medications.
- Medication doses do not need alteration and no medication should be withheld because of concern of fetal teratogenicity.

Differential diagnosis and decisions:

Identify and treat reversible causes of arrest using the acronym (BEAU-CHOPS):

- Bleeding/DIC
- Embolism: Coronary/pulmonary/amniotic fluid embolism
- Anesthetic complications
- Uterine atony
- Cardiac disease [myocardial infarction (MI)/ischemia/aortic dissection/cardiomyopathy]
- Hypertension/pre-eclampsia/eclampsia
- Other differential diagnosis of standard ACLS guidelines (6 Hs and 6 Ts)
- Placenta abruptio/previa
- Sepsis.

Decision for Perimortem Cesarean Delivery

The purposes of perimortem CS are first to facilitate resuscitation and second early delivery of the baby and thus decrease the risk of permanent neurological damage from anoxia. Studies have shown that singleton pregnancy of >20 weeks can also cause significant aortocaval compression. Hence, it is recommended that perimortem cesarean delivery (PMCD) should begin at 4 minutes to effect delivery at 5 minutes after failed resuscitative

efforts. However, the providers need not wait 5 minutes for obvious nonsurvivable injury, especially if the fetus is viable. One needs to remember that the woman should not be transported to the operating room to perform PMCD and need not await the arrival of a surgical instrument to perform PMCD; only a scalpel is adequate.

Q. What factors influence the chances of an infant's survival?

Ans.

- Short interval between the mother's arrest and the infant's delivery
- No sustained prearrest hypoxia in the mother
- Minimal or no signs of fetal distress before the mother's cardiac arrest
- Aggressive and effective resuscitative efforts for the mother
- Hysterotomy is performed in a medical center with an NICU.

SUGGESTED READING

1. American Heart Association. 2005 American Heart Association (AHA) guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiovascular care (ECC) of pediatric and neonatal patients: pediatric basic life support. *Pediatrics*. 2006;117(5):e989-1004.
2. Cant NE, Kenneth J, Leveno Md, Larry C, Gilstrap, Md, John C, et al. In: Cary Cunningham F (Ed). *Williams Obstetrics*, 21st edition. New York: McGraw-Hill Professional; 2001.
3. Fun-Sun F, Yao F, Malhotra V, Fontes ML. Yao and Artusio's *Anesthesiology: Problem-oriented Patient Management*, 6th edition. Philadelphia: Lippincott Williams & Wilkins; 2007.
4. Habib AS. What's new in obstetric anesthesia? The 2017 Gerard W. Ostheimer lecture. *Anesth Analg*. 2019;129(1):168-75.
5. Kosci A. Controlling the hemodynamic response to laryngoscopy and endotracheal intubation. *J Clin Anesth*. 1996;8:63-79.
6. Lindheimer MD, Taler SJ, Cunningham FG. AAOS guidelines 2008 for treatment of local anesthetic toxicity. *American Society of Hypertension. ASH position paper: hypertension in pregnancy. J Clin Hypertens (Greenwich)*. 2009;11:214-25.
7. Lindheimer MD, Taler SJ, Cunningham FG. Hypertension in pregnancy. *J Am Soc Hypertens*. 2010;4:68-78.
8. Miller RD. *Anesthesia*, 6th edition. Philadelphia, PA: Elsevier Churchill Livingstone; 2005.
9. NICE guideline. (2019). Intrapartum care for women with existing medical conditions or obstetric complications and their babies. [online] Available from: <https://www.nice.org.uk/guidance/ng121/resources/intrapartum-care-for-women-with-existing-medical-conditions-or-obstetric-complications-and-their-babies-pdf-66141653845957>. [Last accessed February, 2020]
10. Pandya SH. Labour analgesia: recent advances. *Indian J Anesth*. 2010;54:400-8.
11. Royal College of Obstetricians and Gynaecologists. Hypertension in pregnancy; Management of hypertensive disorder in pregnancy—NICE clinical guidelines. *Obstet Gynaecol*. 2010, revised reprint Jan 2011.
12. Update *Anesth*. 2019;34.

Pregnancy: Physiological Changes and Anemia

Reshma Ambulkar, Sumitra Ganesh Bakshi, Madhavi Desai

A 25-year-old woman in her fourth pregnancy presents with fatigue, dizziness, and palpitations. What would be your differential diagnosis?

- Anemia
- Arrhythmias
- Anxiety disorders
- Pregnancy-induced hypertension (PIH).

Q. What are the causes of anemia of pregnancy?

Ans. In India incidence of anemia in pregnancy is as high as 40–80%. The common causes are discussed below:

Physiological anemia of pregnancy: Blood volume begins to increase in the 6th week of gestation and by the end of pregnancy it reaches approximately 40–50% more than in the prepregnant state. Red cell mass increases as much as 20–30% above prepregnancy levels. The plasma volume increase is greater than the increase in red blood cell mass leading to hemodilution resulting in “physiological anemia of pregnancy.”

Acquired causes: There are several other etiologies of anemia but the most common being nutritional deficiency in India. Nutritional deficiency may be of iron or folic acid/vitamin B₁₂.

Iron deficiency anemia (60%): It is the most common type of anemia in pregnancy. Iron stores not adequate to meet the demands of pregnancy. The body is unable to keep up with the blood volume expansion in the second and third trimesters resulting in iron deficiency anemia. This can be prevented by encouraging pregnant women to supplement their diet with 60 mg/day of elemental iron.

Macrocytic anemia (10%): This is due to deficiency of folic acid and/or vitamin B₁₂. Patients with vitamin B₁₂ deficiency need further workup in addition to serum levels of vitamin B₁₂ to determine the level of intrinsic factor to exclude pernicious anemia.

Folate deficiency in pregnancy is often associated with iron deficiency, though it is less common than iron

deficiency. Folic acid is transported to the placenta by active transport. An increased in mean corpuscular volume (MCV) can be suggestive of folate deficiency. In patients with Crohn's disease and alcoholism, additional folate supplement is required.

Infections: Although rare, anemia can be caused by infections such as parvovirus, cytomegalovirus (CMV), human immunodeficiency virus (HIV), hepatitis viruses, Epstein-Barr virus (EBV), and malaria. If the patient's history suggests exposure to any of these infectious agents, appropriate laboratory studies should be performed.

Blood loss: Blood loss at delivery and postpartum can also cause anemia. The average blood loss with a vaginal birth is about 500 mL and 1,000 mL with a cesarean delivery.

Aplastic or hypoplastic anemia: Pregnancy induces erythroid hypoplasia and in such cases termination of pregnancy may be indicated.

Anemia of chronic disease: Anemia can be a feature associated with chronic disease such as neoplasm, chronic infection (tuberculosis), chronic renal failure, cancer, chemotherapy or HIV infection. During pregnancy, anemia worsens due to the associated medical condition.

HELLP syndrome: It is discussed in the section on Anesthetic Considerations in Patients with Pregnancy-induced Hypertension.

Hereditary: Inherited disorders such as thalassemias, sickle cell anemia, and hereditary spherocytosis can also be the cause of anemia.

withstand the adverse effects of excessive blood loss. Also it leads to an increased risk of infection:

- Poor weight gain
- Preterm labor
- Dysfunctional labor
- Intranatal hemorrhage
- Anesthesia risk
- Cardiac failure
- Postnatal sepsis.

Fetal: There is an increased risk of low birth weight due to poor fetal growth and preterm birth.

- Poor Apgar score
- Fetal distress requiring prolonged resuscitation.

Neonatal and infant:

- Anemia due to poor reserve
- Higher prevalence of failure to thrive
- Poorer intellectual developmental milestones
- Cardiovascular morbidities.

Q. What are the indications for blood transfusion in pregnancy?

Transfusion in pregnancy carries additional risks of RBC alloimmunization, volume overload, and fetal hemolytic disease. Therefore, blood should be transfused judiciously in the antepartum period. Transfusion is indicated if Hb is 4 g/dL in pregnancy <36 week, with or without signs symptoms of anemia or when Hb is between 5 and 7 g/dL in pregnancy <36 weeks, with presence of impending heart failure, hemodynamic instability or acute hemorrhage. When the pregnancy is >36 weeks, blood transfusion is indicated in a woman with Hb < 7 g/dL even without signs of cardiac failure or hypoxia. During labor and postpartum period, blood transfusion is indicated in a woman with severe anemia with decompensation or acute hemorrhage with decompensation with signs of hemodynamic instability.

Q. What are the risk factors for anemia in pregnancy?

Ans. Risk factors for anemia in pregnancy are:

- **Sociodemographic factors:** Age, level of formal education, marital status, areas of residence.
- **Obstetrical factors:** Gravidity, parity, and multiple pregnancies.
- **Behavioral factors:** Smoking, tobacco, and alcohol usage.
- **Medical conditions:** Diabetes, renal or cardiorespiratory diseases, tuberculosis (TB).

Q. How would a patient with anemia in pregnancy present?

Ans.

- Asymptomatic
- Vague complaints of ill health

- Fatigue
- Loss of appetite
- Dyspnea
- Palpitation.

Clinical examination may reveal pallor, pale nails, koilonychia, and pale tongue.

Q. How would you investigate a patient presenting with anemia?

Ans.

Hemoglobin estimation: Despite limitation, it is a useful method for detecting anemia.

Study of peripheral smear:

- **Iron deficiency anemia:** The peripheral smear looks pale; there is hypochromia (large central vacuoles), and microcytosis (small deformed red cells), and anisocytosis (varying size of RBCs).
- **Megaloblastic anemia:** There is macrocytosis (MCV is >95), and fully hemoglobinized red blood cells. In addition to macrocytes, oval macrocytes and hypersegmented neutrophils are seen in various types of macrocytic anemia. When the anemia is more severe, there may be marked poikilocytosis, with teardrop poikilocytes and red-cell fragments.
- **Normochromic normocytic anemia** is seen in patients with chronic diseases such as long-standing infections, TB, malignancy, and renal failure. In hemolytic anemia there would be polychromatic cells, stippled cells, and target cells. In patients with sickle cell disease peripheral smear will reveal sickle cells and large number of reticulocytes.
- **Microspherocytes** (cells that are both hyperchromic and significantly reduced in size and therefore in diameter) may be present in low numbers in patients with a spherocytic hemolytic anemia but are also characteristic of burns and of microangiopathic hemolytic anemia.

Following are special laboratory investigations which may be required for establishing the cause of anemia:

- Total iron-binding capacity (TIBC)
- Serum ferritin concentration (SF)
- Serum folic acid levels
- Serum iron concentration
- Bone marrow studies
- Sick test
- Hb electrophoresis.

The serum iron, total iron-binding capacity, and serum ferritin are the best indicators of iron available for erythropoiesis.

Q. How do you treat iron deficiency anemia?

Ans. Response to iron therapy can be documented by an increase in reticulocytes 5–10 days after the initiation of

Definition

Anemia is defined as hemoglobin (Hb) value <2 standard deviations below the mean value for a healthy matched population. The various definitions of anemia in pregnancy are as follows:

Reference	Definition of anemia	Comment
World Health Organization, 2011	Hemoglobin (Hb) < 11.0 g/dL	Does not define different Hb values as cut off for anemia during different trimesters. Hb values may fall by 5 g/dL during the second trimester
British Committee for Standards in Haematology, 2012	<11.0 g/dL in the first trimester <10.5 g/dL in the second and third trimesters <10.0 g/dL in the postpartum period	In view of the relative plasma expansion leading to hemodilution particularly in the second trimester, it would be reasonable to consider 10.5 g/dL as the cut off from 12 weeks
American College of Obstetricians and Gynaecologists, 2008	<11.0 g/dL in the first trimester <10.5 g/dL in the second and third trimesters <11.0 g/dL in the postpartum period	Based on collected data from European studies of healthy pregnant women
Australian Patient Blood Management Guideline, 2015	There is no agreed normal range for hemoglobin values in pregnant women in Australia. Higher Hb levels may also be associated with adverse pregnancy outcomes, hemoglobin levels between 10.3 and 14.6 g/dL are assumed to be safe during normal pregnancy.	

Q. Why is the incidence of anemia in pregnancy in India very high?

Ans. In India, many people are vegetarian and therefore have vitamin B₁₂ deficiency. In addition to it, the Indian subcontinent has a high content of phytates in their staple diet. The content of inositol hexaphosphate (phytate) and inositol pentaphosphate which are known inhibitors of iron absorption are high in chapati.

Q. How can one prevent anemia in pregnancy?

Ans. India was the first developing country to take up a National Program to prevent anemia among pregnant women and children. The National Anemia Prophylaxis Program of iron and folic acid distribution to all pregnant women in India through the primary healthcare system was evolved and implemented from 1972.

Ensuring good nutrition, balanced diet and supplementation can prevent anemia in pregnant women.

The diet and supplementation should include the following:

- **Iron-rich foods:** Pulses, cereals, jaggery, beet root, green leafy vegetables, meat, liver, egg, fish, legumes, dry beans, and iron-enriched white breads.
- **Iron supplements:** National Nutritional Anemia Prophylaxis Program advises 60–120 mg of elemental iron.
- **Folic acid supplements:** Women who are pregnant and breastfeeding should take 500 µg–1 mg of folic acid a day. It is one of the most important nutrients to have during pregnancy and before conception, as women deficient in folic acid have a greater risk of having a baby with a neural tube defect such as spina bifida.
- **Folic acid rich foods:** Green leafy vegetables, citrus and kiwi fruit, peas, beans, cauliflower, fortified breakfast cereals, fortified bread. Several observational and controlled trials have shown that neural tube defects can be reduced by 80% or more when folic acid supplementation is started before conception.
- **Vitamin B₁₂ supplements:** Vitamin B₁₂ supplement required is 3 µg/day. Treatment of vitamin B₁₂ deficiency includes 0.1 mg/day for 1 week, followed by 6 weeks of continued therapy to reach a total administration of 2 mg.
- **Vitamin B₁₂ rich food:** Meat, egg, milk.

Severity of Anemia

Anemia is defined as a qualitative or quantitative deficiency of Hb. It can be classified as follows:

- | | |
|----------------------|---------------------|
| • Mild anemia | Hemoglobin 9–11 g% |
| • Moderate anemia | Hemoglobin 7–9 g% |
| • Severe anemia | Hemoglobin 4–7 g% |
| • Very severe anemia | Hemoglobin < 4 g% |

Q. What are the compensatory mechanisms in anemia?

Ans.

- Increase in cardiac output
- Rightward shift of oxygen dissociation curve
- Decrease in blood viscosity
- Increase in 2,3-diphosphoglycerate (2,3-DPG) concentration in red blood cells
- Release of renal erythropoietin to stimulate erythroid precursors in bone marrow.

The tissue oxygenation is not impaired in physiological and chronic anemia due to compensatory mechanisms but sudden onset of severe anemia may precipitate cardiac failure and pulmonary edema.

Q. What are causes of concern in anemia associated with pregnancy?

Ans. Anemia is a known risk factor for many maternal and fetal complications.

Maternal: Severe anemia is associated with a higher mortality rate and may be related to a poor ability to

therapy. The Hb concentration increases about 1 g/dL weekly.

Oral iron is safe, inexpensive, and effective way to administer iron. Oral route should be the route of choice in routine cases. Ferrous sulfate is least expensive and allows more elemental iron absorbed per gram administered. Oral iron must be continued for 3–6 months after Hb has come to normal levels in postpartum period.

Parenteral route of iron therapy: The indications for parenteral iron therapy are:

- Patient unable to tolerate oral iron
- Patients suffering from inflammatory bowel disease
- Noncompliant patient
- Patient near term
- Failure of oral therapy.

Parenteral iron has the advantage of causing less gastrointestinal discomfort and inconvenience. Intramuscular iron should be administered in upper outer quadrant of buttock using a Z-track technique. Intravenous iron is expensive and has greater morbidity than oral preparations of iron. Intravenous route should be reserved for those who do not wish to have frequent intramuscular injections.

Iron-sorbitol-citric acid complex (75 mg) is used for intramuscular route only.

Iron-dextran (each mL contains 50 mg of elemental Fe) can be used both by intramuscular and intravenous route, it is available as 50 mg/mL and in 2 mL and 1 mL vials. The administration is done as follows:

- **Test dose:** A test dose (25 mg Fe) is required for first exposure. This should be diluted in 50–100 mL of normal saline (NS) and infused over 15–20 minutes. Monitor heart rate (HR), respiration rate (RR), and blood pressure (BP) every 15 minutes for 1–2 hours after test dose is started.
- It can either be administered as one large dose (diluted in 500–1,000 mL NS and infused over 4–8 hours) or divided into many smaller doses given 1–3 times per week.
- Each 100 mg dose may be administered undiluted as IV push.
- Alternatively each 100 mg dose is diluted in 250 mL NS and infused over 30–60 minutes.

Iron sucrose is given as an infusion. It is available as 5 mL vials of 20 mg/mL strength:

- Each 5 mL contains 100 mg (20 mg/mL) of elemental iron as iron sucrose.
- No test dose is required.
- Each 5 mL vial may be diluted in a maximum 100 mL.
- It is diluted in normal saline and administered over at least 20 minutes.

Iron III carboxymaltose is a ferric hydroxide carbohydrate complex, which allows for controlled delivery of iron within the cells of the reticuloendothelial system, primarily bone marrow and subsequent delivery to the iron-binding proteins ferritin and transferrin. It is administered intravenously, as a single dose of 1,000 mg over 15 minutes (maximum 15 mg/kg by injection or 20 mg/kg by infusion).

Iron III isomaltoside (Monofer): There is little data on its use in the obstetrics.

Total dose infusion (TDI): Iron can be given intravenously at one shot as TDI. Utmost caution is needed for total dose of iron therapy via intravenous route because of severe anaphylactic reaction that may occur. Therefore, total dose of iron therapy by intravenous route should only be given in a hospital setting where facilities are available to manage severe reaction.

The total dose is calculated as follows:

Total dose of iron in mg (for TDI) = $(15 - \text{patients Hb\%}) \times \text{body weight in kg} \times 3$.

Nephritis, cardiorespiratory disease, and allergy are contraindications to parenteral iron therapy.

Blood/packed cell transfusions: Transfusion of packed RBC is reserved for patients with either significant acute bleeding or patients in danger of hypoxia and/or coronary insufficiency or anemia discovered at term.

Q. What are the side effects/complications of parenteral iron therapy?

Ans.

- Muscular injections can cause pain and staining of the skin at injection site, myalgia, arthralgia, and injection abscess.
- Intravenous administration can cause pain in the vein, flushing, and metallic taste in mouth.
- **Allergic reactions:** With intravenous iron therapy reactions can range from rash to anaphylaxis and death. More common with iron-dextran preparation.
- Abdominal pain with nausea and vomiting.
- Headache, dizziness, disorientation, seizures.
- Wheezing, dyspnea, respiratory arrest.
- Chest pain, tachycardia, vascular collapse.
- Iron toxicity presents with nausea, dizziness, and a sudden drop in BP. It is more common with the iron-dextran preparation.

Q. What are the important principles while anesthetizing an anemic patient?

Ans. Tissue oxygenation in anemia is hindered by the low oxygen-carrying capacity of the blood. However in most cases, compensatory changes occur which tend to restore the balance (Available oxygen = Cardiac output $\times$ Arterial oxygen content). In pregnant patient as the cardiovascular

system gets overstretched due to already elevated cardiac output, HR, and increased myocardial work, it can drive the pregnant patient to high output cardiac failure.

In a pregnant woman, severe desaturation due to reduced oxygen-carrying capacity is very likely (excess demand and decreased supply). Since maternal oxygenation directly affects fetal oxygenation, it is important that meticulous attention is paid during the conduct of labor analgesia/regional or general anesthesia to prevent any harm to the baby. Neonatologist skilled in resuscitation forms an important part of the team during delivery.

Q. How does anemia affect oxygen-carrying capacity of the blood?

Ans. Oxygen-carrying capacity of blood is calculated as:

$$CaO_2 = (Hb) \times 1.34 \times (\%SaO_2/100) + (0.003 \times PaO_2).$$

One gram of Hb binds to 1.34 mL of O_2 . In blood 97% of oxygen is bound to Hb, and only 3% is dissolved in plasma, which decides the PaO_2 . 3 mL of oxygen is dissolved in 1 L of plasma. Normally, if Hb is considered as 15 g/dL, then oxygen-carrying capacity is 19–20 mL/100 mL of blood at SaO_2 97–100%. But if patient's saturation is 100% but Hb is 5 g/dL, then patient's oxygen-carrying capacity of blood will be 6.7 mL/100 mL.

Q. What is the preoperative transfusion trigger in anemic patient?

Ans. Healthy myocardium can compensate for low Hb (7–8 g/dL) and hematocrit (21–24%). But transfusion is definitely indicated for Hb < 6 g/dL and rarely indicated for Hb > 10 g/dL in a stable patient. For a woman in labor or in postpartum period, the decision of transfusion is based on patient's clinical condition, ongoing blood loss, existing comorbidities if the Hb ranges from 7 to 9 g/dL.

Q. Discuss preoperative considerations in an anemic pregnant patient.

Ans. In the assessment of the pregnant anemic patient for anesthesia and surgery, the cause of the anemia is of prime importance. Unexpected anemia should be investigated and treated. Appropriate oral or parenteral therapy with iron, vitamin B_{12} or folic acid takes some time and is thus practical only in patients requiring purely elective surgery which can be deferred to a reasonable time period. Recombinant erythropoietin can also be considered in the treatment but its role is yet to be established.

In contrast, when emergency surgery is required, there is little time to diagnose or treat the anemia beforehand. Transfusion of whole blood or packed red cells would raise the total circulating volume but it should be done at least 24 hours prior to surgery to allow restoration of 2,3-DPG levels.

Q. What are the anesthetic goals in an anemic pregnant patient?

Ans.

- Minimize blood loss while maintaining tissue oxygenation
- Good IV access for maintenance of intravascular volume
- Regional anesthesia wherever possible
- Careful positioning to reduce venous pressure
- Scrupulous surgical technique
- Adequate FiO_2 to maintain SpO_2
- Maintenance of adequate oxygen-carrying capacity by using blood transfusion if needed.

Q. Discuss induction and maintenance of anesthesia in an anemic pregnant patient.

Ans.

- *Adequate preoxygenation is essential:* Functional residual capacity (FRC) is decreased, oxygen-carrying capacity is reduced, and the apnea time could also be prolonged in intubation in a pregnant woman which may result in rapid and significant fall in PaO_2 .
- *Use of apneic oxygenation during intubation:* The allowable apneic time before patient starts desaturating can be prolonged using insufflations of oxygen through nostrils using oxygen flow of minimum 10 L/min via nasal prongs.
- *Minimize the reduction in cardiac output:* Titrated doses of induction agents known to have little effect on myocardial contractility.
- Avoid hyperventilation, hypovolemia, hypothermia, acidosis and peripheral pooling of blood due to adverse posturing.
- Optimal blood replacement with goals of transfusion to achieve Hb > 7–8 g/dL (or HbA > 40% of the total Hb in case of abnormal Hb).
- Avoid or vigorously treat the conditions which increase oxygen demand as in shivering or fever.
- Nitrous oxide should be used with caution in patients with folate and vitamin B_{12} deficiency.

Q. Discuss postoperative care in an anemic pregnant patient.

Ans. The aim is again to maximize oxygen delivery and minimize blood loss. Patients should be closely monitored (hemodynamics) in high dependency unit (HDU). Provide oxygen supplementation, maintain euvolemia, good pain relief, and treat any infection aggressively.

Q. What cardiovascular changes occur during pregnancy? What are the implications of these changes?

Ans. Cardiovascular changes begin as early as from 4th week of gestation and are to maximize oxygen transport to placenta. These changes are caused by increased levels

of circulating estrogen and progesterone, which cause vasodilatation and a consequent fall in peripheral vascular resistance by 20%. This causes a fall in systolic and diastolic BP and a reflex increase in HR by 15%. Stroke volume is increased by 25% and together with HR causes an increase of cardiac output by 50% by the third trimester. Myocardial thickness as well as volume of chambers increases resulting in left heart enlargement. Flow murmurs are quite common due to increase plasma volume and cardiac output. Cardiac output may further increase during labor and in immediate postdelivery period on account of autotransfusion. Even during this period there may not be substantial increase in central venous pressure (CVP) or pulmonary capillary wedge pressure (PCWP) due to the vasodilatory effect of progesterone on arterioles and veins. The increased blood volume serves two purposes. First, it facilitates maternal and fetal exchanges of respiratory gases, nutrients and metabolites. Second, it reduces the impact of maternal blood loss at delivery. Anatomically the heart is displaced upward and to the left by the gravid uterus. **Table 1** summarizes the differences in cardiovascular parameters between a pregnant and a nonpregnant woman.

TABLE 1: Differences in cardiovascular parameters between pregnant and nonpregnant woman.

Parameter	Pregnant	Nonpregnant
Heart rate (bpm)	>80	70
Cardiac output (L/min)	6.0	4.5
SVR (dyne.s.cm ⁻⁵)	1,200	1,400
PVR (dyne.s.cm ⁻⁵)	70–80	120
CVP (cm H ₂ O)	5–6	5–6
Colloid oncotic pressure (mm)	16–18	22

(CVP: central venous pressure; PVR: pulmonary vascular resistance; SVR: systemic vascular resistance).

The clinical implications of these changes are:

- In pregnant patient with heart disease and low cardiac reserve, the increase in the work of the heart may cause ventricular failure and pulmonary edema. In these women, further increase in cardiac workload during labor must be prevented by effective pain relief, optimally provided by extradural analgesia. Since cardiac output is highest in the immediate postpartum period, sympathetic blockade should be maintained for few hours after delivery and then weaned off slowly.
- **Heart size and displacement:** The ECG reflects these changes including left axis deviation, ST segment depression and T wave flattening.

Q. What is aortocaval compression? What is its significance?

Ans. From mid-pregnancy, the enlarged uterus compresses both the inferior vena cava and the lower aorta when

the patient lies supine. Obstruction of the inferior vena cava reduces venous return to the heart leading to a fall in cardiac output by as much as 20% toward term. In the unanesthetized state, most women are capable of compensating for the resultant decrease in stroke volume by increasing systemic vascular resistance and HR. There are also alternative venous pathways, the paravertebral and azygos systems. During anesthesia, however, these compensatory mechanisms are reduced or abolished so that significant hypotension may develop rapidly. Obstruction of the lower aorta and its branches causes diminished blood flow to kidneys, uteroplacental unit and lower extremities.

By 38–40 weeks gestational age, there is a 25–30% decrease in cardiac output (CO) when turning from the lateral to the supine position. As the uteroplacental circulation possesses no autoregulation properties, this causes a decreased uterine blood flow and reduced placental perfusion.

Clinical implications: No woman in late pregnancy or labor should lie supine without shifting the uterus off the great abdominopelvic vessels. During cesarean section and for other indications demanding the supine position, the uterus should be displaced, usually to the left, by placing a rigid wedge under the right hip and/or tilting the table left side down. During regional anesthesia, the effects of aortocaval compression will be exaggerated due to a lack of compensatory reflexes subsequent to the sympathetic blockade. This can lead to profound hypotension. In extreme hypotension (or fetal compromise such as a bradycardia), the patient can be turned to the full left lateral position.

Q. What respiratory system changes occur during pregnancy? What is their significance to the anesthetist?

Ans. Respiratory changes occurring during pregnancy are to meet the increased requirement of oxygen as the oxygen consumption nearly doubles at term. There is an increase in the respiratory rate as well as the tidal volume. This also results in low PaCO₂ and higher PaO₂. There is reduction in FRC. Chest wall compliance and lung compliance decrease by about 25–30%.

Anatomical dead space may decrease due to airway edema especially in patients with PIH. **Table 2** differentiates the respiratory system in a pregnant and nonpregnant woman.

Clinical Implications

- Pregnant patients are more prone to hypoxia. Increased oxygen consumption and the decreased reserve due to the reduced functional residual capacity may result in rapid falls in arterial oxygen tension despite careful maternal positioning. This is more marked in obese

patients and with multiple pregnancies. Further the reduced functional residual capacity causes airway closure in 50% of parturients at term in the supine position making preoxygenation less effective.

TABLE 2: Respiratory system changes in a pregnant and nonpregnant woman.

Parameter	Pregnant	Nonpregnant
RR (per min)	16	12–14
Tidal volume (mL)	650	450
Vital capacity (mL)	3,200	3,200
Expiratory reserve volume (mL)	500	700
Inspiratory reserve volume (mL)	2,050	2,050
FRC (mL)	1,300	1,600
Residual volume (mL)	800	1,000
pH	7.44	7.4
PaCO ₂ (mm Hg)	30	35
PaO ₂ (mm Hg)	100–110	90–100

(FRC: functional residual capacity; RR: respiration rate).

- Preoxygenation is essential due to rapid desaturation.
- *Rapid induction with inhalation agents:* The increased minute ventilation combined with decreased functional residual capacity hastens inhalation induction or changes in depth of anesthesia. This is due to increased tidal volume which results in large volume of inspired gas mixing with smaller volume of alveolar gas.
- Reduced compliance necessitates higher airway pressures to maintain adequate ventilation.

Q. Why is gravid considered to be a “full stomach” patient?

Ans. Heartburn during pregnancy is very common and as many as 80% suffer from reflux at term, aggravated by the supine position. The gastrointestinal system is considered to return to normal 24–48 hours postpartum. The effect of progesterone results in reduction of lower esophageal sphincter pressure. Later in pregnancy, the mechanical effects of gravid uterus cause an increase in intragastric pressure and a decrease in gastroesophageal angle. Placental gastrin secretion is increased which worsens gastric acidity. All these changes put the mother at increased risk of aspiration.

Prevention: Prophylaxis in the form of a H₂ blocking drug, nonparticulate sodium citrate and metoclopramide to all pregnant patients for surgery from second trimester onward is a must. The danger of aspiration is almost eliminated when regional anesthesia is used. During general anesthesia airway protection by means of a cuffed endotracheal tube is mandatory, so is rapid sequence induction from second trimester of pregnancy till 48 hours postpartum. Extubation

should be done with the patient awake and on their side to reduce the risk of aspiration of gastric contents.

Q. Why is a pregnant woman said to be living in a state of “accelerated starvation”?

Ans. Metabolic functions are increased during pregnancy to provide for the demands of fetus, placenta, and uterus as well as for the Gravid's increased basal metabolic rate and oxygen consumption. Protein metabolism is enhanced to supply substrate for maternal and fetal growth. Fat metabolism increases as evidenced by elevation in all lipid fractions in the blood. Carbohydrate metabolism, however, demonstrates the most dramatic changes to meet the growing demands of the fetus. Hence, metabolically speaking, pregnant woman live in a state of “accelerated starvation.”

Q. What are the hematological changes seen in pregnancy?

Ans. During pregnancy the plasma volume increases by 45%. This increase is mediated by a direct action of progesterone and estrogen on the kidney causing the release of renin and thus an activation of the aldosterone-renin-angiotensin axis. This leads to renal sodium retention and an increase in total body water. Due to an increase in renal erythropoietin production, red cell mass increases by 20%. As the increase in red cell mass is relatively smaller than that of plasma volume, the Hb falls from 14 g/dL prepregnancy to 12 g/dL during the third trimester. The blood viscosity is reduced which may slightly decrease cardiac work. At 2 weeks postpartum, the blood volume returns to prepregnancy levels. **Table 3** summarizes these changes.

The white cell count rises throughout pregnancy and peaks after delivery. Pregnancy is a hypercoagulable state. Plasma concentrations of fibrinogen and all clotting factors, except XI and XIII gradually increase. Although there is an increase in platelet production, the platelet count falls because of increased platelet activity and consumption. Platelet function remains normal in pregnancy. The changes in the coagulation system are detailed in **Table 4**.

TABLE 3: Hematological changes in pregnant and nonpregnant woman.

Parameter	Pregnant	Nonpregnant
Hematocrit	0.30–0.34	0.4
Hemoglobin (g/dL)	11–12	13–14
RBC ($\times 10^{12}/L$)	3.8	4.2
ESR	30–70	10
WBC ($\times 10^9/L$)	9	6
Plasma volume (mL/kg)	80–85	65

(ESR: erythrocyte sedimentation rate).

TABLE 4: Changes in coagulation system.

Coagulation factor/parameter	Changes
Fibrinogen	Increased from 2.5 to 5 g/L
Factor II	Slightly increased
Factor V	Slightly increased
Factor VII	Increased 10-folds
Factor VIII	Increased twofold
Factors IX and X	Increased
Factor XI	Decreased 70%
Factor XII	Increased 40%
Factor XIII	Decreased 40%
Bleeding time	WNL
PT	WNL
PTT	WNL

(PT: prothrombin time; PTT: partial thromboplastin time).

Clinical Implications

- The increased circulating volume offers protection for mother and fetus from the effects of hemorrhage at delivery but it can delay the onset of the classical signs and symptoms of hypovolemia.
- Raised WBC count makes a diagnosis of infection more difficult. There is also an increase in ESR.
- Thromboembolic complications remain a common source of morbidity and mortality associated with pregnancy.
- In preeclampsia there is an increase in thromboxane A₂ produced by platelets which causes vasoconstriction and decreases uteroplacental blood flow.

Q. Why is failed intubation more common during pregnancy?

Ans.

- During pregnancy, there is capillary engorgement and edema of the upper airway down to the pharynx, false cords, glottis, and arytenoids due to fluid retention. Edema of the airway makes upper airway obstruction and bleeding more likely and may make tracheal intubation more difficult.
- When airways of pregnant patients were examined at 12 and 38 weeks of gestation, in 34% of the pregnant women the Samsoon modification of the Mallampati classification worsens to grade IV, which may further worsen during the labor. Therefore, it is imperative to examine the airway immediately before cesarean delivery, rather than relying solely on information from prelabor evaluation.
- The increase in chest diameter and enlarged breasts can make laryngoscopy with a standard Macintosh blade difficult. Failure to intubate the trachea is seven

times more common in the term parturient compared to nonpregnant patients.

- Anticipate difficult intubation and a need for a range of ETT sizes also airway adjuncts should be readily available. A smaller diameter endotracheal tube may be required for intubation especially in cases of preeclampsia.

Q. Which other systems are affected during pregnancy and what are the implications?

Ans. Renal: Renal plasma flow and glomerular filtration rate (50%) increase during pregnancy. Blood urea and serum creatinine are reduced by 40%. 24 hours creatinine clearance is also increased. Renin-angiotensin, aldosterone and progesterone are increased leading to water retention and a decreased plasma osmolality. The tubular reabsorption of glucose falls and glycosuria is present in 40% of parturients. Progesterone-mediated ureteric smooth muscle relaxation can lead to urinary stasis making pregnant women prone to urinary tract infections.

Clinical implications: There is an increase in the volume of distribution for drugs and may have to be given in higher than normal dosages.

Hepatic: Liver blood flow does not increase. Plasma cholinesterase falls by 25%. The levels of alanine transaminase (ALT), aspartate transaminase (AST), and lactate dehydrogenase (LDH) are slightly elevated. Plasma concentrations of alkaline phosphatase are increased threefold as a result of placental production. Serum albumin is decreased reducing the plasma oncotic pressure.

Clinical implications: There may be a prolongation of neuromuscular blockade after administration of succinylcholine. This is not usually clinically significant. The total concentration of plasma protein is reduced due to the increase in plasma volume. This results in a drop in the colloid oncotic pressure, and may account for the edema seen in pregnancy. Also the pharmacokinetics of protein bound drugs will be affected.

Endocrine: There is an increased insulin resistance during pregnancy. The placenta produces human placental lactogen which has similar effects to growth hormone and may be the cause of maternal insulin resistance. Any carbohydrate load will cause a higher than normal increase in plasma glucose concentrations.

Clinical implications: The thyroid gland undergoes hypertrophy during pregnancy. Increased production of thyroxine and triiodothyronine are normally balanced by increased production of thyroid binding globulin so the levels of the free hormones remain the same. Levels of parathyroid hormone tend to fall during pregnancy as does

the level of serum calcium, although the level of ionized calcium remains constant.

Musculoskeletal system: The placenta produces relaxin which causes relaxation of ligaments. This results in widening and increased mobility of the pubis and sacroiliac joints to allow passage of the fetus through the birth canal. Due to the enlarging uterus, there is a compensatory increase in the lumbar lordosis. As a result, backache is a common complaint during pregnancy.

Clinical implication: Due to increased lumbar lordosis, subarachnoid block or epidural become more challenging.

Others: Blood flow to nasal mucosa increased.

Clinical implications: Nasal intubation is frequently associated with epistaxis.

Blood flow to the skin is increased resulting in warm and clammy feet. This is to dissipate heat from the metabolically active uteroplacental unit.

Q. What are the neurological changes in a pregnant woman? Discuss the implications to the anesthetist.

Ans. The minimal alveolar concentration (MAC) of volatile anesthetics decreases during pregnancy. This may be secondary to the high levels of progesterone and possibly an increase in B endorphin levels. There is a similar increase in sensitivity to opioids, sedatives and local anesthetics. The effects of local anesthetic drugs when used for neuraxial anesthesia and analgesia are also enhanced secondary to mechanical factors within the epidural and subarachnoid space. Compression of the inferior vena cava results in diversion of blood through the vertebral venous plexus that lies within the epidural space. This causes the epidural veins to engorge and the volume of the epidural and subarachnoid space to decrease. The pressure of the cerebrospinal fluid (CSF) is increased due to compression from the epidural veins in the epidural space. Between contractions the pressure may be around 28 mm Hg but during painful contractions it may rise to as much as 70 mm Hg. This altered response, subsides progressively in the early postpartum period.

Clinical Implications

- As the volume of the epidural and subarachnoid space decreases, an identical volume of local anesthetic will spread more extensively in the pregnant than in the nonpregnant state. Increased pressure in space facilitates diffusion of drug into dural space. Venous congestion of lateral foramina decreases loss of local anesthetic along the dural nerves. During the last months of gestation, approximately two-thirds of the normal dose is adequate.

- As the CSF pressure rises during contraction, it is probably safer not to advance an epidural or spinal needle during contractions for risk of puncturing the dura and expulsion of CSF at high pressure.
- Also epidural veins are dilated by the action of progesterone and cannulation of an epidural vein when performing epidural insertion ("a bloody tap") is also more common.

Q. What are functions of placenta and what factors affect the transfer of drugs from maternal to fetal side?

Ans. The placenta is a vital barrier and link between maternal and fetal circulations. The barrier between fetal and maternal circulations is two-cell thick, fetal capillary endothelium, and it is covering syncytial trophoblast. It is an imperfect barrier and substances do cross from the mother to fetus.

Functions

- **Gas exchange:** O₂ and CO₂ exchange.
- **Hormonal synthesis:** Placenta synthesizes and releases estrogen, progesterone, chorionic gonadotropin, prolactin, prostaglandins.
- **Nutrient supply:** Water, electrolytes, minerals.
- **Detoxification** of drugs and substances.
- **Transfer of immunity** from mother to fetus by way of immunoglobulins.

Placental transport mechanisms are:

- **Simple diffusion:** O₂ and CO₂ transport occurs due to the difference between partial pressures on both sides. Fatty acids are also transported by means of simple diffusion.
- **Secondary active transport:** Amino acids are transferred mostly as linked carriers. Sodium down its concentration gradient drags amino acids with it.
- **Pinocytosis:** Placenta is impermeable to proteins, only immunoglobulin G (IgG) is transported.
- **Facilitated diffusion:** Glucose.
- **Bulk transport:** Water and electrolytes.

Factors affecting placental transfer of drugs are:

- **Lipid solubility:** The placental membrane is freely permeable to lipid soluble substances which undergo flow-dependent transfer. Higher the lipid solubility higher the transfer of drugs.
- **Molecular weight:** Smaller molecular weight drugs diffuse easily (<600 Da).
- **Degree of ionization:** Ionized form will not cross the barrier easily. The degree of ionization of acidic drugs is greater on the maternal side and lower on the fetal side. The converse applies to basic drugs.
- **Protein binding:** Protein bound drugs will not diffuse easily, only free drug would cross the placental barrier easily. Acidosis reduces the protein binding of local

anesthetic. Reduced albumin concentration increases the proportion of unbound drug.

- **Maternal fetal concentration gradient:** The rate of transfer is governed by Fick law of diffusion, when the transfer is by simple diffusion. *Note:* Fetal-maternal concentration is used as an index for transfer of drugs.

Anesthetic Drugs

- **Opioids:** All opioids cross the placenta in significant amounts. They are weak bases, bound to α -1 glycoprotein.
- **Pethidine:** Longer half-life is due to its active metabolite norpethidine, which may lead to respiratory depression in the neonate.
- **Morphine** is poor lipid soluble but readily crosses the placenta due to low protein binding and increased free drug.
- **Fentanyl:** It is highly lipid soluble and albumin bound, so crosses the placental barrier easily.
- **IV induction agents:** Sodium thiopentone is highly lipid soluble, weakly acidic, 75% protein bound and <50% ionized at physiological pH. It crosses the placenta easily with a F/M ratio of 0.4–1.1. Doses of >8 mg/kg produce neonatal depression, whereas lower doses produce no significant neonatal effects providing induction to delivery time is <5 minutes.
- **Propofol:** It is highly protein bound and lipophilic. F/M ratio of 0.6–0.8.
- **Inhalational agents:** These agents are highly lipid soluble with low molecular weights.
- **Muscle relaxants:** These are quaternary ammonium compounds and fully ionized. These drugs are fully ionized as well as have low lipid solubility; hence they do not cross the placenta.
- **Local anesthetics:** These drugs have low molecular weights (<600 Da) and also are lipid soluble. Different drugs have different protein binding.
- **Bupivacaine** because of its high protein binding (96%) crosses the placenta in lesser concentration than

lignocaine (65%). Transfer to the fetus also is dependent on the site of administration (paracervical route concentration will be higher than epidural).

- **Anticholinergics:** Atropine passes through the placental barrier unlike glycopyrrolate.
- **Benzodiazepines:** Diazepam crosses the placenta easily (highly lipid soluble and nonionized). F/M ratio is 1 as compared to midazolam with F/M ratio 0.76.

SUGGESTED READING

1. Aitkenhead AR, Rowbotham DJ, Moppett I, Thompson J (Eds). Smith and Aitkenhead's Textbook of Anaesthesia, 6th edition. Philadelphia: Elsevier; 2013.
2. Allman KG, Wilson IH. Oxford Handbook of Anaesthesia, 4th edition. UK: Oxford University Press; 2016.
3. Bedson R, Riccoboni A. Physiology of pregnancy: clinical anaesthetic implications. Cont Educ Anaesthesia. Crit Care Pain. 2014;14:69-72.
4. Cunningham GF, Leveno KJ, Bloom SL, Spong CY, Dashe JS, Hoffman BL, Casey BM, Sheffield JS (Eds). William's Textbook of Obstetrics, 24th edition. New York: McGraw Hill Education; 2014.
5. Ferguson MT, Dennis AT. Defining perioperative anaemia in pregnant women—challenging the status quo. Anaesthesia. 2019;74:237-45.
6. Grewal A. Anaemia and pregnancy: anaesthetic implications. Indian J Anaesth. 2010;54:380-6.
7. Kodali BS, Chandrasekhar S, George LN, Topulos P, Datta S. Airway changes during labor and delivery. Anesthesiology. 2008;3:357-62.
8. Muñoz M, Peña-Rosas JP, Robinson S, Milman N, Holzgreve W, Breymann C, et al. Patient blood management in obstetrics: management of anaemia and haematologic deficiencies in pregnancy and in the post-partum period: NATA consensus statement. Transfus Med. 2018;28:22-39.
9. Pavord S, Myers B, Robinson S, Allard S, Strong J, Oppenheimer C. UK guidelines on the management of iron deficiency in pregnancy. London: British Committee for Standards in Haematology; 2011.
10. Tandon R, Jain A, Malhotra P, et al. Management of iron deficiency anemia in pregnancy in India. Indian J Hematol Blood Transfus. 2018;34:204-15.
11. Yao FSE. Yao and Artusio's Anesthesiology, 8th edition. Gurugram: Wolters Kluwer India Pvt. Ltd.; 2016.

Emergency Lower Segment Cesarean Section

Sumitra Ganesh Bakshi, Reshma Ambulkar, Jeson Rajan Doctor

A 22-year-old female with 39 weeks amenorrhea is admitted to labor ward with labor pains. On examination, fetal heart rate is 100/min. Consent for emergency lower segment cesarean section (LSCS) has been taken; patient is to be shifted to the theater. Discuss management.

Q. Discuss fetal monitoring during labor.

Ans. Fetal monitoring begins in the antepartum period. The important consideration in deciding when to begin antepartum testing is based on the threat to neonatal survival and the severity of maternal disease. Normal labor is a process of repeated fetal hypoxic events, which results in acidemia. Normal parturition is an asphyxiating event for the fetus. It is important to monitor fetal well-being during the labor process.

Fetal well-being is dependent on an intact uteroplacental unit. Simultaneous monitoring of fetal heart rate (FHR) and uterine contraction helps in detection of fetal distress. Continuous cardiotocography (CTG) also known as electronic fetal monitoring is the most commonly performed obstetric procedure.

The National Institute of Child Health and Human Development (NICHD) fetal monitoring workshop classifies the normal FHR patterns as follows: Baseline heart rate: 110–160 beats per minute (bpm) with variability of 6–25 bpm with absence of any decelerations. If the baseline FHR is <110 bpm, it is termed bradycardia. If the baseline rate is >160 bpm, it is termed tachycardia.

Beat-to-beat variability is an index of cardiovascular function and is regulated by the cardiovascular system, such irregularity is defined as baseline variability. Abnormalities in FHR, such as bradycardia, tachycardia, dysrhythmias, decreased or absent beat-to-beat variability or transient decelerations can be a sign of fetal asphyxia. Transient or periodic decelerations in FHR fall into the following three categories depending upon their time with respect to maternal contractions.

Early, or type 1, decelerations: Decrease in FHR, which occurs simultaneously with the onset, peak and end of

contraction. This is due to the vagal stimulus caused by the fetal head compression and usually not associated with fetal hypoxia.

Late, or type 2, decelerations: A gradual decrease in FHR is seen which begins after the onset of a contraction and lasts beyond the end of a contraction. This is suggestive of uteroplacental insufficiency and requires prompt attention.

Variable, or type 3, decelerations: Changes in FHR, which is unrelated to uterine contractions. They usually result from umbilical cord compression. They are further classified as:

- *Mild:* FHR 80 bpm lasting < 30 seconds, usually insignificant
- *Moderate:* FHR < 70 bpm for 30–60 seconds, signify mild hypoxia
- *Severe:* FHR < 70 bpm lasting > 60 seconds, indicate fetal acidosis.

If the FHR tracing is normal, 99% of the time the fetus is not depressed. In contrast, if FHR tracing is abnormal in 50% cases, the infants are normal with no umbilical cord acidosis. Fetal scalp capillary blood pH, FHR accelerations and fetal pulse oximetry are additional methods to assess fetal well-being.

Q. What are the causes of fetal distress?

Ans. Causes of fetal distress includes:

- Maternal hypotension
- Placental abruption
- Umbilical cord compression (secondary to cord prolapse/oligohydramnios)
- Pregnancy-induced hypertension
- Uterine hypertonus.

Q. What are the implications of meconium-stained amniotic fluid?

Ans. Three theories have been suggested to explain fetal passage of meconium:

1. The pathological explanation proposes that the fetus passes meconium in response to hypoxia, and that meconium signals fetal compromise.
2. In utero passage of meconium may represent normal gastrointestinal maturation under neural control.
3. Meconium passage could also follow vagal stimulation from common but transient umbilical cord entrapment and resultant increased peristalsis.

In conclusion, high incidence of meconium observed in the amniotic fluid during labor often represents fetal passage of gastrointestinal contents in conjunction with normal physiological processes.

When fetal acidemia supervenes, in cases of fetal hypercarbia, the fetal respiration is stimulated leading to aspiration of meconium into the alveoli. This leads to secondary alveolar damage. Thus, meconium in amniotic fluid is an environmental hazard for the fetus.

Q. What do we understand by Mendelson's syndrome?

Ans. Mendelson was the first who described acute chemical aspiration pneumonitis. Patients are considered at risk for aspiration pneumonitis if gastric volume exceeds 25 mL (0.4 mL/kg) and pH is <2.5.

Early signs include:

- Cyanosis
- Tachycardia
- Massive pulmonary edema
- Bronchospasm occurs often (unlike with amniotic fluid embolism)
- Hypotension
- Hypovolemia with hemoconcentration (the reactive transudation of fluid into the lungs contributes to this finding).

Later cardiac failure may develop and accompanying this. There may be:

- Increased pulmonary artery pressure
- Reduced static lung compliance
- Falling arterial oxygen
- Severe metabolic acidosis (usually develops later)
- Infection is not usually a feature
- Chest X-ray shows pulmonary edema and patchy atelectasis (but there is often poor correlation between the extent of pulmonary damage and the radiological appearance).

Differential Diagnosis

- Amniotic fluid embolism
- Pulmonary embolus (see amniotic fluid embolism)

- Other causes of shock and circulatory collapse include:
 - Abruptio placentae
 - Heart disease
 - Other pulmonary diseases including asthma, pneumothorax
 - Subarachnoid hemorrhage
 - Malignant hyperthermia—very rare.

Q. How will you prevent Mendelson's syndrome? Or what preoperative medications would you give this group of patients?

Ans. Prevention of Mendelson's syndrome: Preventive measure may be applied in labor (particularly in patients at risk of having a cesarean section), before cesarean section and postpartum (e.g. with anesthesia for retained placenta) and includes avoidance of general anesthesia where possible, and use of regional anesthesia, epidural or spinal anesthesia.

Aspiration pharmacoprophylaxis:

- Nonparticulate antacid, 30 mL of 0.3 molar (M) sodium citrate. This has duration of action of 40–60 minutes.
- Intravenous (IV) administration of histamine receptor (H_2) blocking agents 40 minutes before airway management. Omeprazole, a proton-pump inhibitor and antisecretory agent require 40 minutes to reduce gastric acidity. Ranitidine is preferred. H_2 -receptor antagonists should be repeated every 6 hours.
- Administration of metoclopramide decreases gastric volume as a prokinetic agent within 30–60 minutes. It crosses the placenta but has not been shown to have any lasting adverse neurobehavioral effects on the newborn.

In case of urgent surgery, the above drugs should nevertheless be administered for reducing the risk of aspiration at extubation.

Other measures include, good anesthetic technique including:

- Presence of experienced obstetric anesthetist at every cesarean section or anesthetic for retained placenta.
- Preoxygenation of the patient before intubation. Use of Sellick's maneuver (cricoid pressure) to prevent regurgitation.
- Identifying patients likely to be difficult to intubate. Patients can be identified according to certain characteristics (short neck, etc.) but also if intubation has been difficult with previous anesthetics.
- Compliance with and training in a "failed intubation procedure".

Q. Would you insert a nasogastric or orogastric tube preoperatively?

Ans. The use of a nasogastric or orogastric tube prior to induction of anesthesia, to enable physical emptying of the stomach has been suggested; particularly if a parturient has

had opiates in labor and is therefore most likely to have a full stomach.

Physical removal of gastric contents would reduce the volume by a greater degree than pharmacological methods, but the remaining volume may still make the pregnant woman vulnerable to aspiration.

The efficacy of cricoid pressure in presence of gastric tube is controversial. Hence, the gastric tube could be pulled out prior to induction. There exists another school of thought that in a parturient at high risk of aspiration, it may be more practical to insert a nasogastric or orogastric tube after induction of anesthesia, once the airway is secured. As the risk of aspiration continues into the recovery period, this may be a logical approach and one that is more acceptable to the mother.

Q. What do you understand by Sellick's maneuver (cricoid pressure)?

Ans. Cricoid pressure, sometimes called Sellick's maneuver (or even the Sellick's), is the application of backward pressure on the cricoid cartilage to occlude the esophagus.

Cricoid pressure should be performed during induction of anesthesia for both emergency surgery (full stomach) and for elective surgery when lower esophageal sphincter is likely to be incompetent (e.g., last half of pregnancy or gastroesophageal reflux disease), and in patients with delayed gastric emptying (e.g., diabetic autonomic neuropathy).

Q. How do you perform Sellick's maneuver?

Ans. Locate the most prominent protuberance on the front of the neck in the midline (the thyroid prominence). Find this point then run your finger toward the patients' feet (staying in the midline) until you feel your finger drop into the cricothyroid notch or membrane. The next horizontal bar is the cricoid cartilage. Place the thumb and index finger on either side of the cricoid cartilage and press directly backward at a force of 20–30 Newtons (N) against the cervical vertebrae, which is about 2–3 kg (10 N = ~1 kg). Maintain pressure until directed to release. The application of cricoid pressure requires a dedicated rescuer as it must be maintained until the airway is secured by endotracheal intubation. This may be difficult where there are limited rescuers or when prolonged application is required. While too little force is ineffective, overzealous efforts can restrict ventilation and may worsen laryngoscopic view.

Cricoid pressure should not be confused with optimal external laryngeal manipulation (OELM) or backward upward right pressure (BURP) on the thyroid cartilage, which is used to improve visualization of the vocal cords when intubating. BURP is performed by an assistant and moves the larynx to the right while the tongue is displaced

to left by the laryngoscope blade. These are techniques employed to improve visualization of the cords, and do not protect the lungs from regurgitation.

Cricoid pressure trainer: It encompasses realistic patient anatomy with real-time feedback on the technique.

Cricoid pressure and the laryngeal mask airway (LMA): Cricoid pressure has been shown to prevent gastric insufflations when used with LMA.

Although the LMA can be successfully inserted while cricoid pressure is being applied, some have stated it makes insertion difficult, and others found ventilation may be impeded. If insertion of LMA fails, cricoid pressure may be momentarily released and reapplied after successful insertion.

Cricoid Pressure and Pediatrics

The application of cricoid pressure in the pediatric population has been shown to prevent insufflations of gas into stomach during face mask ventilation.

Q. How would you manage if aspiration occurs?

Ans.

- Tilt the operating table to a 30° head low to prevent further aspiration and allow gastric contents to drain to outside
- Maintain cricoid pressure
- Suck the mouth and pharynx as rapidly as possible
- Intubate the patient with immediate inflation of endotracheal cuff to prevent aspiration
- Suction through endotracheal tube before administering 100% oxygen
- Insert orogastric tube to empty the stomach, measure pH of gastric contents
- Tracheobronchial aspirate collected for culture and sensitivity
- Electively ventilate
- Treat bronchospasm
- Serial arterial blood gas (ABG), watch for hypoxia
- Early application of positive end-expiratory pressure (PEEP).

Q. Is there any role of prophylactic antibiotic and steroids in aspiration?

Ans. There is no role of prophylactic antibiotic; excluding feculent aspirate, which can occur in obstructed cases—antimicrobial therapy for anaerobic and gram-negative organisms may be warranted. Superinfection can occur. Cultures should be collected and treat as clinically indicated. The role of steroid is controversial. Pneumonia caused by gram-negative bacteria was more frequent after aspiration in patients treated with corticosteroids than in those who were not. The benefit of high-dose corticosteroid

in patients with acute respiratory distress syndrome (ARDS) is debatable and routine administration of steroids cannot be recommended for ARDS.

In acid aspiration pneumonitis, pulmonary lesions were aggravated by irrigation of bronchial tree as the large volume of fluid helped to push the acid deeper and mixing of acid and treatment solution. Bronchial irrigation is indicated only in obstructive type of aspiration. 5–10 mL of normal saline is instilled into the tracheobronchial tree, followed immediately by suction. This is to be repeated till aspirate fluid is clear.

Q. What anesthetic technique would you use for emergency lower segment cesarean section (LSCS)?

Ans. The choice depends on:

- Indications for the surgery
- The degree of urgency
- Maternal status.

Q. How do you classify urgency of LSCS?

Ans. The classification of urgency of LSCS has been shown in Table 1.

TABLE 1: Classification of urgency of lower segment cesarean section.

Grade	Definition
I: Emergency	Immediate threat to life of woman or fetus
II: Urgent	Maternal or fetal compromise which is not immediately life-threatening
III: Scheduled	Needing early delivery but no maternal or fetal compromise
IV: Elective	At a time to suit the woman and maternity team

Q. Discuss advantages and disadvantages of various anesthetic techniques.

Ans.

Spinal Anesthesia

Advantages:

- Rapid onset
- Provides dense block
- Small dose of local anesthetic is used, so there is minimal transfer of drug to fetus
- Failures are very infrequent with block
- Decreased risk of failed intubation and aspiration of gastric contents
- Avoidance of depressant agents
- Ability of mother to remain awake and enjoy the birthing experience
- It has been suggested that blood loss is reduced under regional anesthesia.

Disadvantages:

- Higher incidence of hypotension
- Finite duration of anesthesia

- Despite achieving an adequate (T4) block, some women under spinal anesthesia will experience some degree of visceral discomfort during the surgery (particularly in situation in which the obstetrician exteriorizes the uterus). The quality can be improved by adding opioids
- Postdural puncture headache (PDPH) with larger needles
- Total spinal block—a rare and serious complication that occurs after excessive cephalad spread of the local anesthetic.

Hematoma: The incidence of neurologic injury resulting from hematoma associated with spinal is very low with estimates of 1 in 220,000.

Epidural Anesthesia

Advantages:

- It provides flexibility, when catheter is placed for labor analgesia and can be used for section.
- The volume of local anesthetic drug can be titrated.
- Incidence of hypotension is less.
- All advantages of regional block as mentioned above.

Disadvantages:

- Takes a little longer time for insertion and institution of analgesia.
- Large doses of local anesthetic are used to achieve adequate levels.
- Failures including incomplete or patchy block are more frequent than spinal.

General Anesthesia

It is necessary when:

- Life-threatening fetal compromise is present
- Cases with overt coagulopathy
- Maternal hemorrhage.

Advantages:

- Speed of induction
- Control of airway
- Superior hemodynamics.

Q. What is rapid sequence spinal anesthesia? Discuss.

Ans. It is a new approach of spinal anesthesia for emergency LSCS. It aims in reducing the induction time for spinal anesthesia, which has been the deciding factor in choosing between spinal and general anesthesia in emergency cases. The aim of rapid sequence is to establish spinal anesthesia with the bare essentials and to abandon the same in case of failure of the block, thus also limiting the number of attempts. For rapid spinal anesthesia, it is suggested to use sterile gloves but no gown. The position for spinal anesthesia would be based on obstetric factors and anesthetist preference. Lateral position is preferred in

most places as it is best for the uterine blood flow in cases of fetal compromise. The risk of pain or conversion to general anesthetic, in case of failure, is much lower than the risk associated by giving all these patients straight away a general anesthetic. If needed, surgery can be paused after delivery, to allow anesthesia to intensify.

Q. What is dural puncture epidural (DPE) technique?

Ans. The DPE is similar to a needle-through-needle combined spinal epidural technique. In this technique after puncturing the dura with the spinal needle, no intrathecal medications are given, and all medications are administered through the epidural catheter. The dural puncture is believed to be the conduit for drug spread. Initial results suggest early onset of block with this technique. Cerebrospinal fluid return through the spinal needle passing through the epidural can further serve as a confirmatory test for correct positioning of the epidural needle tip within the epidural space. Currently, DPE appears to be a favorable technique; however, finer details and limitations of this technique are still under evaluation.

Q. Can neuraxial technique be used for any other obstetric interventions?

Ans. In the absence of contraindications, the American College of Obstetricians and Gynecologists recommends offering external cephalic version to all near-term women with breech presentations under central neuraxial blockade.

A meta-analysis reported that neuraxial techniques for external cephalic version increase the success rate by 44% and reduce the cesarean delivery rate by 17%, while also reducing pain scores during the procedure.

Q. How would you treat spinal hypotension?

Ans.

- Coload with crystalloids means giving IV fluids concurrently during and after giving neuraxial local anesthetic.
- Preloading means giving IV crystalloids before giving neuraxial block.
- Use of compression stocking prior to administration of neuraxial blockade limits venous pooling.
- Left uterine displacement with the help of a wedge beneath the right buttock and thus reducing aortocaval compression by the gravid uterus.
- *Use of vasopressor:* Phenylephrine is preferred as few studies have shown less fetal acidosis with the use of phenylephrine when compared to ephedrine.

Q. If you had unexpectedly been unable to intubate this patient, how would you manage the airway?

Ans. Flowchart 1 shows the management of airway.

Q. What are the salient features of the All India Difficult Airway Association (AIDAA) 2016 guidelines for the management of unanticipated difficult tracheal intubation in obstetrics?

Ans.

Step 1: Laryngoscopy and tracheal intubation

If unable to intubate at first attempt with direct/video laryngoscopy during modified rapid sequence induction—call for help:

- Continue nasal oxygen using O₂ flow at 15 L/min
- One more attempt at intubation [only if oxygen saturation (SpO₂) ≥ 95%]
- Mask ventilation between attempts using gentle intermittent positive pressure ventilation (IPPV) with the adjustable pressure limiting (APL) valve closed to ≤20 cm H₂O
- Partial/complete release of cricoid pressure if mask ventilation is inadequate
- Optimize position, partial/complete release of cricoid pressure, external laryngeal manipulation to optimize view and use bougie/stylet, if required
- Consider changing device/technique/operator between attempts
- Maintain depth of anesthesia
If successful, check position of endotracheal tube using capnography.
If unsuccessful, move to Step 2.

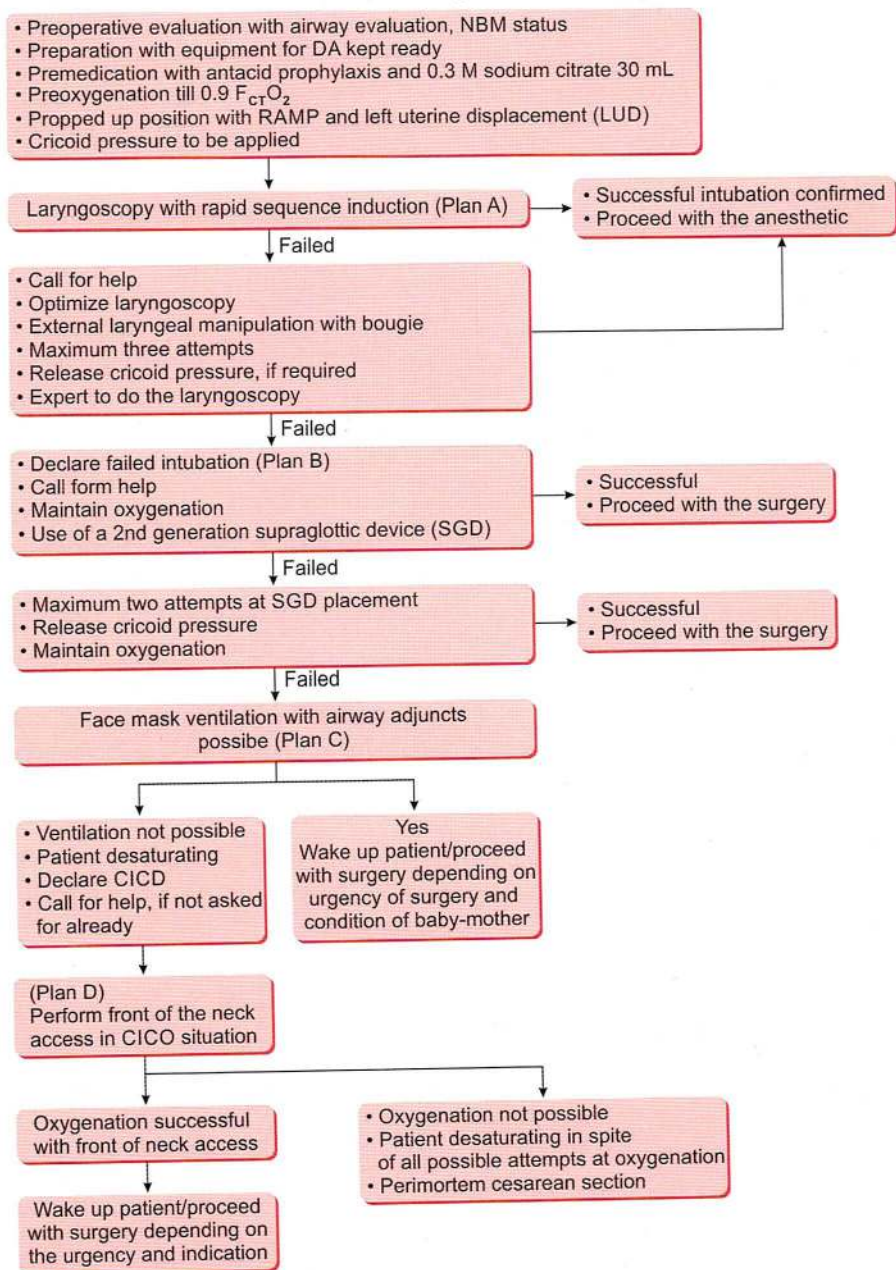
Step 2: Supraglottic airway device (SAD) insertion to maintain oxygenation:

- Continue nasal oxygen using O₂ flow at 15 L/min
- Preferably use second-generation SAD
- Remove cricoid pressure during insertion
- Maximum two attempts (only if SpO₂ ≥ 95%)
- Consider changing size or type of SAD
- Maintain depth of anesthesia
If successful, the options are to continue anesthesia using a SAD, intubate through the SAD with a fiberoptic scope if expertise and equipment is available. Consider awakening the mother if fetal and maternal conditions are stable.
If mother has been awakened, proceed under central neuraxial block or general anesthesia following awake fiberoptic intubation.
If unsuccessful, move to Step 3.

Step 3: Rescue face mask ventilation:

- Continue nasal oxygen using O₂ flow at 15 L/min
- Ensure neuromuscular blockade
- Final attempt at face mask ventilation using optimal technique and adjuncts

Flowchart 1: Management of airway.



If successful and it is an emergency surgery continue with rescue face mask ventilation. Consider awakening the mother if fetal and maternal conditions are stable and feasible.

If mother has been awakened, proceed under central neuraxial block or general anesthesia following awake fiberoptic intubation.

If unsuccessful, call for additional help and declare complete ventilation failure (CVF) and proceed to Step 4.

Step 4: Emergency cricothyrotomy:

- Continue nasal oxygen using O₂ flow at 15 L/min and efforts at rescue face mask ventilation
- Perform one of the following techniques:

- Surgical cricothyroidotomy
- Wide-bore cannula cricothyroidotomy
- Needle cricothyroidotomy (use pressure regulated jet ventilation and attempt to keep the upper airway patent).

If successful and it is an emergency surgery continue with rescue face mask ventilation. Consider awakening the mother if fetal and maternal conditions are stable and feasible.

If unsuccessful and the situation deteriorates into maternal cardiac arrest continue advanced life support and perform perimortem cesarean delivery within 4 minutes of cardiac arrest.

Q. What is the effect of inhalation agent on uterine contractility?

Ans. If less than one minimum alveolar concentration (MAC) of a potent volatile inhalation agent, such as isoflurane or sevoflurane is used, there is no increase in blood loss or decrease in uterine contractility. At these low concentrations, the uterus still responds to oxytocin. At higher concentration, uterine contractility is decreased and blood loss is increased.

Q. What is Apgar scoring system? What is its significance?

Ans. Apgar score is a useful aid to evaluate the need for infant resuscitation applied at 1 and 5 minutes after birth. The score was devised in 1952 by Dr Virginia Apgar. An Apgar score of 10 is in practice rarely assigned. Most infants are in excellent condition at birth and have Apgar score of 7–10 (Table 2).

TABLE 2: Apgar scoring system.

Mnemonic	Sign	Score		
		0	1	2
A	Appearance (color)	Blue, pale	Body pink, extremities blue	Pink
P	Pulse	Absent	<100/min	>100/min
G	Grimace (reflex irritability; response to catheter in nose)	Absent	Grimace	Cough, sneeze
A	Activity (muscle tone)	Limp	Some extremity flexion	Active motion
R	Respiratory effort (breathing)	Absent	Weak or irregular	Strong efforts with a good cry

A low 1-minute Apgar score does not correlate with the infant's future outcome. Change in score between 1 and 5

minutes is a useful index of effectiveness of resuscitation efforts. Apgar scores at 1 and 5 minutes correlate poorly with either cause or outcome. Correlation of the Apgar score with future neurological outcome increases when the score remains 0 to 3 at 10, 15 and 20 minutes.

Q. Discuss the immediate resuscitation of the newborn before the neonatologist arrives.

Ans. Those newly born infants who do not require resuscitation can generally be identified by a rapid assessment of the following four characteristics:

1. Was the baby born after a full-term gestation?
2. Is the amniotic fluid clear of meconium and evidence of infection?
3. Is the baby breathing or crying?
4. Does the baby have good muscle tone?

If the answer to all four of these questions is "yes", the baby does not need resuscitation and should not be separated from the mother. The baby can be dried, placed directly on the mother's chest, and covered with dry linen to maintain temperature. Observation of breathing, activity, and color should be ongoing. If the answer to any of these assessment questions is "no", there is general agreement that the infant should receive one or more of the following four categories of action in sequence:

1. Initial steps in stabilization (provide warmth, position, clear airway, dry, stimulate, reposition)
2. Ventilation
3. Chest compressions
4. Administration of epinephrine and/or volume expansion.

For additional details, refer to cardiopulmonary cerebral resuscitation (CPR) guidelines.

Q. How would you manage postoperative pain?

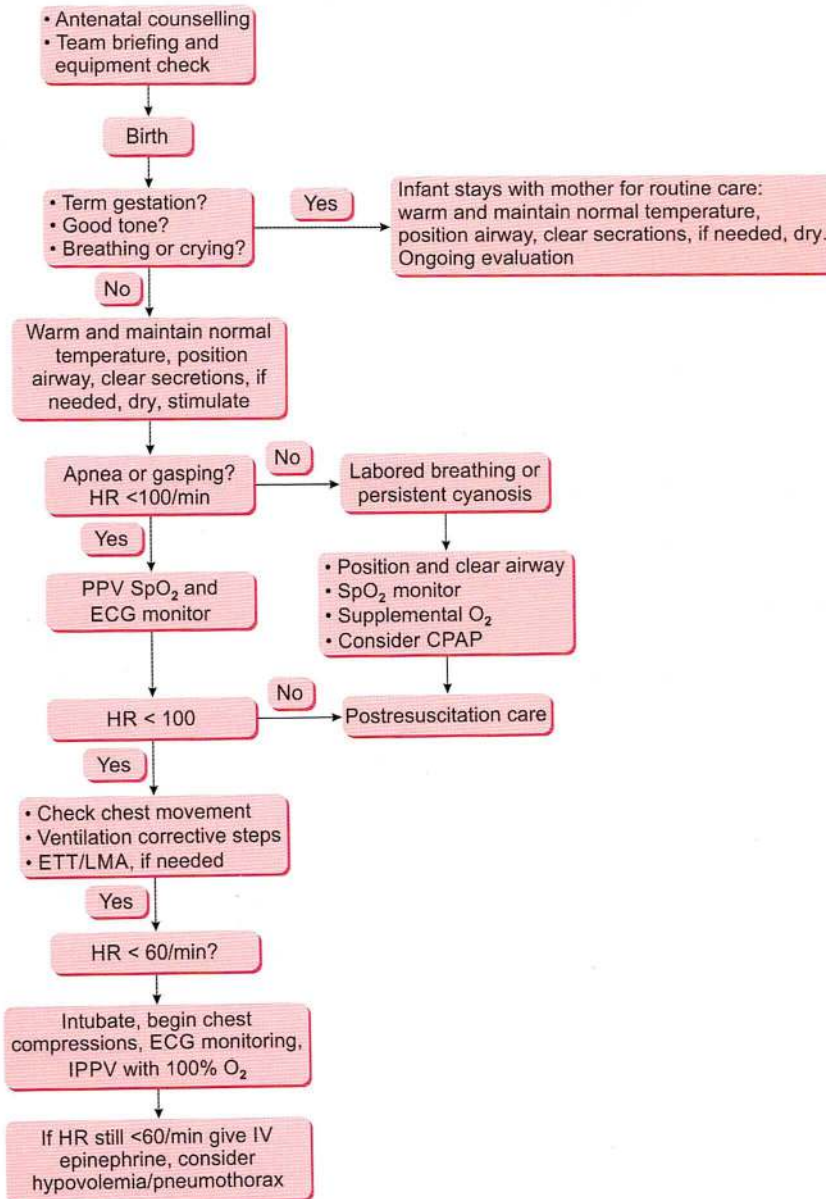
Ans. As per guidelines laid by Breastfeeding Network. Paracetamol is safe and can be given in dose of 1 g four times a day.

Transfer of nonsteroidal anti-inflammatory drugs (NSAIDs) is small and can be used for pain relief.

Aspirin should preferably be avoided as a painkiller, as the small amounts that are secreted in the milk can cause increased risk of Reye's syndrome in pediatric viral infections. There are reports of neonatal apnea following codeine given to the mother. Hence, the neonate needs to be watched, if the mother is on opioids for pain relief.

Q. Is there any protocol for neonatal resuscitation?

Ans. **Flowchart 2** shows the neonatal resuscitation algorithm 2015 update.

Flowchart 2: Neonatal resuscitation algorithm 2015 update.

SUGGESTED READING

1. Cunningham FG, Gant NF, Leveno KJ, Gilstrap III LC, Hauth JC, Wenstrom KD (Eds). William's Obstetrics, 21st edition. New York: McGraw-Hill; 2001.
2. Habib AS. What's new in obstetric anesthesia? The 2017 Gerard W. Ostheimer Lecture. *Anesth Analg*. 2019;129:168-75.
3. Hien C, Owen H. *J Emerg Prim Health Care*. 2005;3(1-2).
4. Ramkumar V, Dinesh E, Shetty SR, Shah A, Kundra P, Das S, et al. All India Difficult Airway Association 2016 guidelines for the management of unanticipated difficult tracheal intubation in obstetrics. *Indian J Anaesth*. 2016;60:899-905.
5. Rudra A. Airway management in obstetrics. *Indian J Anaesth*. 2005;49(4):328-35.
6. Vasdev GM, Harrison BA, Keegan MT, Burkle CM. Management of the difficult and failed airway in obstetric anesthesia. *J Anesth*. 2008;22:38-48.
7. Yao FF, Malhotra V, Fontes ML (Eds). Yao and Artusio's Anesthesiology: Problem-Oriented Patient Management, 6th edition. Philadelphia: Lippincott Williams and Wilkins; 2007.

Nonobstetric Surgery in a Pregnant Patient

Sumitra Ganesh Bakshi, Reshma Ambulkar, Malini Joshi

A 29-year-old female with previous bad obstetric history is admitted for threatened abortion. Cervical cerclage is planned. Discuss the anesthesia plan.

Q. What is the incidence of surgery during pregnancy?

Ans. About 2% of pregnant women undergo nonobstetric surgery annually.

The operations include:

- Those directly related to pregnancy, such as cervical cerclage.
- Those indirectly related to pregnancy, such as ovarian cystectomy.
- Unrelated to gestation, such as appendectomy.

Q. What is the etiology of early pregnancy loss? What is cervical cerclage?

Ans. The etiology of early pregnancy loss is varied and often controversial. More than one etiologic factor is often present. The most common causes of recurrent miscarriages are as follows:

- *Genetic causes:*
 - Mendelian disorders
 - Multifactorial disorders
 - Chromosomal inversions
- Autoimmune causes
- *Anatomical variations:*
 - Uterine Müllerian anomaly
 - Incompetent cervix
 - Leiomyomas
 - Uterine polyps
- Infectious causes
- *Environmental causes:*
 - Smoking
 - Excessive alcohol consumption
 - Endocrine factors
 - Diabetes mellitus
 - Antithyroid antibodies
 - Luteal-phase deficiency
- Hematologic disorders.

The gestational age at the time of the spontaneous abortions can provide clues about the cause. For instance, nearly 70% of spontaneous abortions in the first 12 weeks are due to chromosomal anomalies. However, losses due to cervical incompetence tend to occur after the first trimester. The cervix normally stays tightly closed during pregnancy.

Occasionally, it starts to open early, leading to miscarriage. For some women, this recurs in subsequent pregnancies. This may be due to cervical weakness (incompetence) and the miscarriage usually occurs in the second or early third trimester. Management includes cervical cerclage—a suture (stitch) is taken to keep the cervix closed.

Q. What are the main concerns of surgery in the pregnant patient?

Ans.

- Maternal concerns
- Effect on developing fetus
- Surgical problem itself.

Maternal safety: During pregnancy, maternal anatomic and physiologic changes occur, which have implication on the anesthetic management. Pregnant woman are more prone to hypoxia and hypercapnia. Airway management can be technically difficult. Physiologic compensation for aortocaval compression can be compromised by anesthetic techniques that interfere with sympathetic tone and can result in profound hypotension. Pregnant women are at increased risk for gastric acid aspiration during anesthetic induction or unconscious sedation.

Effect on developing fetus: Fetal loss, fetal asphyxia, teratogenic effects of the anesthetic agents, premature rupture of membranes, and premature labor.

Surgical problem itself: Especially in nonobstetric surgery.

Q. What are the key principles of anesthetic management of the case?

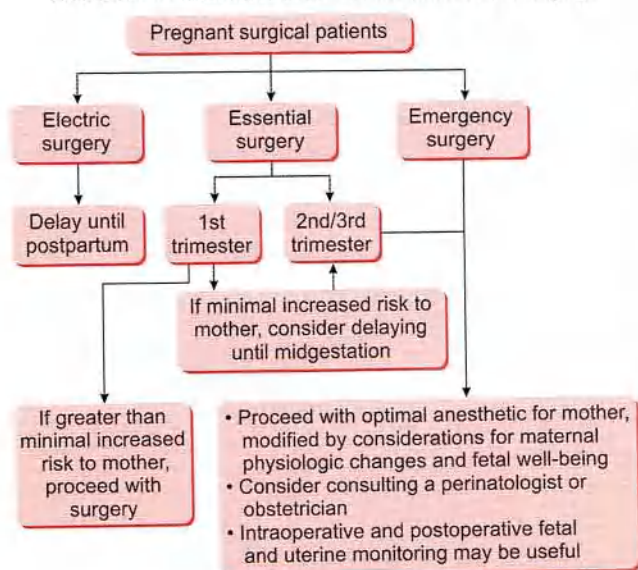
Ans.

- Optimize and maintain normal maternal physiological function.
- Optimize and maintain uteroplacental blood flow and oxygen delivery.
- Avoid unwanted drug effects on the fetus.
- Avoid stimulating the myometrium (oxytocic effects).
- Avoid awareness during general anesthesia.
- Use regional anesthesia, if possible.

Q. What will you discuss with the patient preoperatively?

Ans. The maternal and fetal risk associated with surgery needs to be explained and included in the written consent (Flowchart 1).

Flowchart 1: Maternal and fetal risk associated with surgery.



Q. What are the concerns regarding fetal malformations?

Ans. The most susceptible time for the developing fetus is between 15th and 30th day after conception; susceptibility declines thereafter to the 50th day. Virtually every drug and every inhalation anesthetic is teratogenic to some species under certain conditions. None has as yet been identified as a definite human teratogen. However, we cannot assume that some potential for teratogenicity does not exist. It is therefore most prudent to postpone elective surgical procedures until after pregnancy. If this is not possible then the first trimester should be avoided.

As per US Food and Drug Administration (FDA) fetal risk categories for therapeutic agents are:

- **Category A:** Controlled studies have shown no risk to the fetus during the first trimester and later trimesters as well.

- **Category B:** Animal studies have demonstrated no fetal risk, but no controlled studies have been performed in humans.

- **Category C:** Studies have shown fetal risk in animals (teratogenic or embryocidal) but no controlled human studies.

- **Category D:** Confirmed evidence exists for human fetal risk, but benefits are acceptable despite known risk.

- **Category X:** Agents of this class are contraindicated in pregnant patients for any reason because animal or human studies have displayed teratogenicity or there is evidence of fetal risk from prior human experience.

Most of the anesthetic drugs belong to either category C or B. Following drugs belong to category B: Methohexital, propofol, enflurane, isoflurane, desflurane, sevoflurane, lidocaine, ropivacaine, and fentanyl.

Q. Is nitrous oxide a known teratogen?

Ans. Nitrous oxide oxidizes cobalamin and inhibits methionine synthase activity. This is a key link in the synthesis of S-adenosylmethionine and in the tetrahydrofolic acid cycle. DNA production, myelin deposition, and folate and methylation process-dependent reactions might be affected. Though teratogenicity has not been demonstrated in humans, nitrous oxide is best avoided during the first trimester and if used at other stages of pregnancy, pretreatment of the patient with folic or folinic acid could offer some additional benefit.

Q. How will you premedicate this patient?

Ans. Aspiration prophylaxis should be administered to all patients over 14 weeks' gestation because physiologic changes at lower esophageal sphincter enhance the risk of aspiration. An H_2 antagonist should be given the night prior and 1 hour before surgery if possible and a nonparticulate antacid, such as sodium citrate just before induction of anesthesia. Prokinetic agent, such as metoclopramide 10 mg intravenously enhances gastric emptying. The longstanding relative contraindication and concern about benzodiazepine use, particularly in the first trimester has been dispelled. Appropriate use of benzodiazepine to treat anxiety is essential as catecholamine increased by pain or anxiety may adversely affect uterine blood flow.

Q. How will you decide on the anesthetic technique?

Ans. Anesthetic technique depends upon the clinical status, surgical procedure and patient preferences. Whenever possible, regional anesthesia should be preferred.

Spinal anesthesia: The advantages are that spinal anesthesia offers the least drug transfer, and airway manipulations are not involved. The disadvantages are hypotension, aortocaval compression, and possibility of maternal hypoxia and acidosis (in case of high spinal).

Epidural anesthesia: Regional block yields higher local anesthetic blood levels and hence more placental transfer, however, no teratogenic effect have been seen in humans.

General anesthesia: Though there is better control of airway and hemodynamics there is maximum fetal exposure to anesthetic drugs.

Q. Describe the technique for general anesthesia.

Ans.

- Secure intravenous line.
- Preoxygenate for at least 3 minutes (or 8 vital capacity breaths in 1 minute with oxygen at 15 L/min, end-point exhaled oxygen level > 90%) to ensure denitrogenation and to avoid maternal and fetal hypoxemia during induction and intubation.
- Carry out induction using a rapid sequence technique using thiopentone and succinylcholine.
- Avoid supine position. Displace the uterus laterally; a wedge beneath the right buttock may suffice.
- Maintenance of anesthesia using safe drugs such as opioids, nondepolarizing muscle relaxants, and inhalational agents. Nitrous oxide can be avoided and higher concentration of inhalational agents can be used.
- Avoid hyperventilation and hypoventilation.
- Reversal agents should be administered slowly to avoid acute increase in acetylcholine, which might stimulate uterine contractions.
- Extubate when patient is awake and has regained laryngeal reflexes.

Q. How will you monitor this patient intraoperatively?

Ans. Monitoring should include:

- Electrocardiogram
- Pulse oximetry
- Intermittent blood pressure
- Temperature monitoring
- Capnometer (if general anesthesia planned)—to maintain normocarbia (ETCO₂—30 mm Hg). Higher levels of CO₂ will lead to acidosis, while hypocarbia can reduce uterine blood flow.
- Urine output.

Fetal monitoring should include external fetal heart rate (FHR) monitoring from 18 weeks onward using Doppler ultrasound but whether monitoring can affect outcome is controversial. It is advisable to document FHR before and after institution of both regional and general anesthesia and on completion of surgery. The decision to perform fetal monitoring should be individualized and may be based on gestational age, type of surgery, and facilities available.

Q. What is the role of tocolytic therapy in pregnant patients undergoing nonobstetric surgery? Describe the mechanism of action, side effects in brief.

Ans. There is no proven benefit to routine administration of prophylactic perioperative tocolytic therapy. It may be indicated in select cases with irritable uterine activity.

Indomethacin, a nonspecific cyclo-oxygenase (COX) inhibitor, is the most commonly used tocolytic of this class. It reduces the risk of delivery 48 hours of initiation of treatment. These drugs inhibit prostaglandin synthesis which is essential for the labor process and thus act as tocolytics. Maternal side-effects include gastrointestinal side-effects and platelet dysfunction. Fetal side-effects include premature closure of the ductus arteriosus, bronchopulmonary dysplasia, and intraventricular hemorrhage.

Calcium channel blockers reduce the inflow of Ca⁺⁺ into the cell by inhibiting the slow calcium channels and lead to myometrial relaxation. Relative safety and ease of administration support use of this drug. The maternal side-effects include tachycardia and mild hypotension.

β-agonists act on the specific β₂-receptors and increase intracellular cAMP which reduces myometrial contractility. Some commonly used β-agonist includes ritodrine and terbutaline. Prolonged use of these drugs could be associated with tachyphylaxis. The side effects include tachycardia, arrhythmias, hypotension, pulmonary edema, fetal tachycardia and fetal hypoglycemia.

Less effective drugs include oxytocin receptor antagonists (Atosiban), magnesium sulfate, and progesterone supplementation.

The decision for administration of corticosteroids to the mother for fetal lung maturity would be based depending on the urgency of the surgery, and the obstetrician's estimate of risk of preterm birth. Glucocorticoids are best avoided empirically in the setting of systemic infection, as they may impair the maternal immune system.

Q. Describe postoperative care.

Ans.

- Provide adequate hydration.
- Continue left uterine displacement.
- Administer supplemental oxygen. Consult the obstetrician if tocolytics are needed in the postoperative period, and USG may be repeated to ascertain fetal well-being.
- **Thromboprophylaxis:** Pregnancy is a hypercoagulable state with increases in the majority of coagulation factors and decreases in protein S levels. In all pregnant patients undergoing surgery mechanical devices—pneumatic compression devices should be used. The need to administer pharmacological thromboprophylaxis

should be individualized based on the type of surgery, length of immobilization, and associated high-risk factors such as obesity, previous history of venous thromboembolic (VTE) disease. One should weigh the benefit of VTE prevention as against the risk of bleeding and fetal complications.

Q. Discuss postoperative pain management.

Ans.

- Pain increases risk of premature labor.
- Opioids are safe for the fetus and preferred for postoperative pain relief.
- Regional and field blocks provide good pain relief with opioid sparing action and must be considered whenever feasible.
- Nonsteroidal anti-inflammatory drugs (NSAIDs) are avoided after first trimester because some of agents may close the fetal ductus arteriosus. They are also associated with oligohydramnios with reduced fetal renal function.

■ SUGGESTED READING

1. Cheek TG, Baird E. Anesthesia for non-obstetric surgery: maternal and fetal considerations. *Clin Obstet Gynecol.* 2009;52:535-45.
2. Cunningham FG, Gant NF, Leveno KJ, Larry C (Eds). *William's Obstetrics*, 21st edition. John Wiley and Sons; 2010.
3. Guyatt GH, Akl EA, Crowther M, Gutterman DD, Schünemann HJ, American College of Chest Physicians Antithrombotic Therapy and Prevention of Thrombosis Panel. Executive summary: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. *Chest.* 2012;141:7S-47S.
4. Kuczkowski KM. Nonobstetric surgery during pregnancy: what are the risks of anesthesia? *Obstet Gynecol Surv.* 2004;59:52-6.
5. Reitman E, Flood P. Anaesthetic considerations for non-obstetric surgery during pregnancy. *Br J Anaesth.* 2011;107:i72-8.
6. Rosen MA. Management of anesthesia for the pregnant surgical patient. *Anesthesiology.* 1999;91:1159-63.
7. Van De Velde M, De Buck F. Anesthesia for non-obstetric surgery in the pregnant patient. *Minerva Anesthesiol.* 2007;73:235-40.
8. Vercauteren M, Palit S, Soetens F, Jacquemyn Y, Alahuhta S. Anaesthesiological considerations on tocolytic and uterotonic therapy in obstetrics. *Acta Anaesthesiol Scand.* 2009;53:701-9.
9. Weingart SD, Levitan RM. Preoxygenation and prevention of desaturation during emergency airway management. *Ann Emerg Med.* 2012;59:165-75.
10. Yao FSF, Malhotra V, Fontes ML(Eds). Yao and Artusio's *Anesthesiology. Problem-Oriented Patient Management*, 6th edition. Philadelphia: Lippincott Williams and Wilkins; 2008.

Amniotic Fluid Embolism

Reshma Ambulkar, Sumitra Ganesh Bakshi, Amol Trimbakrao Kothekar

A woman getting labor analgesia gets breathless and then collapses during second stage of labor. She is cyanosed. There is no evidence of bleeding.

Q. What is your differential diagnosis?

Ans.

- Obstetric causes:
 - Acute hemorrhage
 - Amniotic fluid embolism (AFE)
 - Placental abruption
 - Uterine rupture
 - Uterine atony
 - Eclampsia
 - Peripartum cardiomyopathy
- Anesthetic causes:
 - High spinal anesthesia
 - Aspiration
 - Local anesthetic toxicity
- Nonobstetric causes:
 - Pulmonary embolism
 - Air embolism
 - Anaphylaxis
- Sepsis/septic shock
- Intracerebral bleed
- Drug toxicity
- Acute myocardial infarction.

Q. What do you understand by AFE?

Ans. Amniotic fluid embolism is a life-threatening obstetric emergency. It is associated with amniotic fluid, fetal cells, hair, or other debris entering the maternal circulation. It usually presents as sudden, profound, and unexpected maternal collapse associated with hypotension, hypoxemia, and disseminated intravascular coagulation (DIC).

Most cases occur during labor (70%), 19% during cesarean section, and 11% following vaginal delivery.

Q. What are the risk factors for AFE?

Ans. There are no consistent clinical factors which can predict AFE. However, it is frequently associated with the following conditions:

- Advanced maternal age
- Multiparity
- Meconium-stained liquor
- Obstructed labor
- Intrauterine fetal death
- Polyhydramnios
- Tetanic uterine contractions
- Maternal history of allergy or atopy
- Uterine rupture
- Placenta accreta
- Trauma
- Diabetes mellitus
- Operative delivery including cesarean section.

Q. What is the pathophysiology of AFE?

Ans. The pathophysiology of AFE still remains unclear.

It is uncommonly known as “anaphylactoid syndrome of pregnancy”.

Maternal circulation gets exposed to amniotic fluid or debris via ruptured membranes/ruptured cervical/uterine vessels as a result of a pressure gradient from the uterus to veins. This results in hypoxemia, pulmonary vasospasm, cardiac failure or even death.

In 1995, Clarke described it as an immune-mediated process and results in activation of the complement cascade

stimulating endogenous immunomediators, producing a reaction similar to anaphylaxis.

Also described the biphasic response to AFE:

- **Phase 1:** There is a release of biochemical mediators once amniotic fluid and fetal cells gain entry into the maternal circulation which results in pulmonary artery vasospasm leading to pulmonary hypertension. This results in hypoxemia and hypotension due to elevated right ventricular pressures and right ventricular dysfunction. This phase may last up to 30 minutes.
- **Phase 2:** Left ventricular failure and pulmonary edema may occur in patients who survive the initial insult. Biochemical mediators are known to trigger DIC which eventually leads to massive hemorrhage and uterine atony.

Q. What are the clinical features of AFE?

Ans. The clinical features of AFE have been shown in **Table 1**.

TABLE 1: Clinical features of amniotic fluid embolism.

Symptoms	Signs
Dyspnea	Hypotension
Cough	Fetal distress
Headache	Pulmonary edema/ARDS
Chest pain	Cyanosis
	Coagulopathy
	Seizures
	Bronchospasm
	Cardiopulmonary arrest
	Uterine atony

(ARDS: acute respiratory distress syndrome).

Diagnostic Criteria for AFE (Adapted from Stafford et al.)

- Acute hypotension and/or cardiac arrest
- Acute hypoxia diagnosed by dyspnea, cyanosis and/or respiratory arrest
- Subsequent coagulopathy or severe clinical hemorrhage.

The above occurring during labor, cesarean delivery or dilation and evacuation, or within 30 minutes postpartum with no other explanation for the findings are suggestive of AFE.

Q. Enumerate the changes in various systems as a result of AFE.

Ans.

Cardiovascular changes: The hemodynamic response to AFE is biphasic. Initially there is pulmonary hypertension secondary to pulmonary vasoconstriction, which can precipitate right heart failure. Vasoconstriction in the systemic circulation → hypertension. This is followed by left heart failure and hypotension.

Hematological changes: Activation of a consumptive coagulopathy → rise in activated partial thromboplastin

time (APTT) and prothrombin time (PT) with a fall in fibrinogen levels.

Respiratory changes: Hypoxia secondary to two processes (cardiogenic pulmonary edema secondary to left heart failure, inflammatory capillary leak within the pulmonary vasculature leading to noncardiogenic pulmonary edema).

Neurological changes: Hypoxia associated encephalopathy.

Q. How would you treat a patient of AFE?

Ans. Amniotic fluid embolism is a diagnosis of exclusion. High index of suspicion is necessary to diagnose this life-threatening condition. It should be suspected in sudden onset of dyspnea, cardiovascular collapse and DIC. Ongoing resuscitation and early delivery of fetus should be the plan. The management is supportive and symptomatic with the goal of maintaining oxygenation, hemodynamic support, and correction of coagulopathy.

Team approach: Anesthetists, obstetricians, hematologists, and intensivists.

Immediate Management

Resuscitation: The Airway, Breathing, and Circulation (ABC) approach.

Airway and breathing:

- Administer 100% oxygen via a nonrebreathing reservoir face mask.
- Prompt assessment, with control of the airway and ventilation of the lungs with tracheal intubation may be essential.

Circulation:

- Establish intravenous (IV) access with two large-bore cannulas (take blood at the same time for: crossmatch of 6 units, full blood count, and coagulation studies).
- Left lateral tilt/manual uterine displacement.
- Hemodynamic support would include preload optimization and vasopressors.
- Commence fluid resuscitation with crystalloid/colloid to optimize filling.
- Infusion of an inotrope may be required to maintain a mean arterial blood pressure and achieve an adequate urine output.
- An arterial line for continuous blood pressure monitoring is essential, and the use of a noninvasive cardiac output monitor may be helpful.
- Continuously monitor the fetus and early consideration should be given to delivery of baby.
- **Uterine tone:** Maintenance of uterine tone is important and is achieved by pharmacological agents (oxytocin, ergometrine, and prostaglandins such as carboprost and misoprostol).

- **Coagulation:** The development of consumptive coagulopathy may warrant the use of plasma, cryoprecipitate and platelets which are guided by laboratory investigations. Recombinant factor VII should be considered only if bleeding is not controlled with aggressive blood component therapy as this can cause thrombotic complications (thrombosis in major organs can lead to permanent disability, multiorgan failure, and death) in pregnant patients.
- Antifibrinolytics, like epsilon (ϵ)-aminocaproic acid and tranexamic acid, might be helpful but currently evidence is lacking to support its use.

Q. What investigations would be useful?

Ans.

Investigations:

- Complete blood count
- **Coagulation profile:** AFE is associated with DIC in >80% cases
- **Electrocardiogram:** Tachycardia, ST segment and T-wave changes, and findings consistent with right ventricle strain
- **Arterial blood gases:** Hypoxia
- **Chest X-ray:** Pulmonary edema
- Echocardiogram
- Serum tryptase
- Ventilation-perfusion (V/Q) scan.

Q. What are the problems frequently encountered while treating this patient?

Ans.

- Disseminated intravascular coagulation
- Uterine atony.

Q. How would you prognosticate the patient?

Ans. Maternal mortality rate of 20.4% (11–44%) has been described in population-based studies.

Q. How would you manage cardiac arrest in this lady?

Ans. High-quality cardiopulmonary resuscitation in pregnancy should be done. Preparations for emergent

perimortem cesarean delivery should be initiated simultaneously with the initiation of cardiopulmonary resuscitation in the parturient > 23 weeks of gestation for both maternal and fetal indications. Forceps or vacuum-assisted vaginal delivery may be considered in patients in labor.

Q. How would you manage this lady after delivery of the fetus?

Ans.

- **Intensive care monitoring:** High risk at developing—acute respiratory distress syndrome (ARDS), heart failure, DIC
- **Supportive treatment:** Ventilation, inotropic support, hematological support
- Steroids may be useful.

Potential interventions for severe life-threatening cases of AFE:

- Inhaled nitric oxide for pulmonary hypertension leading to right-sided heart failure
- Extracorporeal membrane oxygenation (ECMO) for severe hypoxia and left heart failure
- Intravenous lipid emulsion (Intralipid).

SUGGESTED READING

1. Allman KG, Wilson IH. Oxford Handbook of Anaesthesia, 4th edition. United Kingdom: Oxford University Press; 2016.
2. Benson MD. Current concepts of immunology and diagnosis in amniotic fluid embolism. Clin Dev Immunol. 2012;2012:946576.
3. Dedhia JD, Mushambi MC. Amniotic fluid embolism. Contin Educ Anaesth Crit Care Pain. 2007;7(5):152-6.
4. Gist RS, Stafford IP, Leibowitz AB, Beilin Y. Amniotic fluid embolism. Anesth Analg. 2009;108:1599-602.
5. Society for Maternal-Fetal Medicine (SMFM); Pacheco LD, Saade G, Hankins GD, Clark SL. Amniotic fluid embolism: diagnosis and management. Am J Obstet Gynecol. 2016;215:B16-24.
6. Yao FF. Yao and Artusio's Anesthesiology, 8th edition. Philadelphia: Wolters Kluwer Health; 2016.

Obstetric Hemorrhage

Reshma Ambulkar, Sumitra Ganesh Bakshi, Aparna Chatterjee

You are called to the labor ward to see a 25-year-old lady [gravida 3, para 2 (G3P2)] in labor, who has lost 700 mL of blood per vaginally. She looks pale and sweaty, her pulse is 140/min, regular but thready and her blood pressure (BP) is 100/50 mm Hg.

Q. How would you manage this patient?

Ans.

- Administer 100% oxygen via a nonrebreathing reservoir face mask.
- Establish intravenous (IV) access with two large-bore cannulas [Take blood at the same time for: crossmatching (6 units), complete blood count, and coagulation studies].
- Place the patient in a head down and left lateral tilt position.
- Start fluid resuscitation with crystalloid/colloid.
- Use group-specific or O Rh-negative blood for resuscitation till cross-matched blood is available.
- Set up a fluid warmer, rapid infusion device and (if available) a cell saver.
- Monitor hematocrit, hemoglobin and coagulation at regular intervals.
- *Communication is vital:* Notify theater staff, alert the blood bank and hematologist.
- Joint assessment of the patient with the obstetrician and decision on further plan of action.
- Continuous maternal and fetal monitoring.

If the massive bleeding continues:

- Give fresh frozen plasma (FFP) and cryoprecipitate and platelets according to the coagulation results. Use thromboelastography (TEG)/rotational thromboelastometry (ROTEM), if available.
- Establish and start invasive monitoring.
- Consider recombinant factor VIIa (rFVIIa) (NovoSeven).

Q. How would you assess the degree of blood loss?

Ans. Blood loss can be notoriously difficult to assess in obstetric bleeds. There could be problems detecting the

loss accurately since the early signs of bleeding mimic the normal physiological changes of pregnancy, e.g., dilutional anemia, increased pulse, and respiratory rate. Bleeding may sometimes be concealed and the presence of amniotic fluid makes accurate estimation difficult. Physical signs which would aid the diagnosis are pallor, tachycardia, low blood pressure (beware: blood pressure of healthy women may not drop until significant amount of blood has been lost), delayed capillary refill, skin color, decreased level of consciousness, and oliguria.

Q. What are the likely causes of her bleeding?

Ans.

- Placenta previa
- Placental abruption
- Uterine rupture
- Cervical or vaginal tear
- Coagulation disorders.

Q. Her previous surgery was a lower segment cesarean section (LSCS). Is it relevant?

Ans. Previous cesarean section increases the possibility of placenta previa and uterine rupture in future pregnancies.

Q. Classify causes of hemorrhage related to pregnancy.

Ans.

Early Pregnancy

- Incomplete abortion
- Septic abortion
- Ruptured ectopic.

Antepartum Hemorrhage

- Placenta previa
- Placental abruption

- Uterine rupture
- Trauma.

Postpartum Hemorrhage

- Primary:
 - Uterine atony
 - Retained products of conception
 - Genital tract trauma
 - Abnormally adherent placenta
 - Clotting defects
 - Acute uterine inversion.
- Secondary:
 - Puerperal sepsis
 - Retained products of conception.

Q. Define massive obstetric hemorrhage.

Ans. Massive obstetric hemorrhage is defined as: loss of >1,500 mL of blood; a drop in hemoglobin > 4 g/dL; or acute transfusion requirement of >4 units of blood. Obstetric hemorrhage is divided into antepartum hemorrhage (APH) and postpartum hemorrhage (PPH). Hemorrhage is termed antepartum when bleeding occurs anytime after 24 weeks of gestation but before delivery. PPH is when bleeding occurs after delivery of baby and can be primary (within 24 hours of delivery) or secondary (24 hours to 6 weeks after delivery).

Q. Discuss the causes of obstetric hemorrhage in details.

Ans.

Antepartum Hemorrhage

Placenta previa and abruption are major causes of significant hemorrhage in the third trimester.

Placenta Previa

It is the encroachment of the placenta upon the cervical os (**Fig. 1**):

- *Grade 1:* Low-lying or lateral placenta (in lower uterine segment) that does not reach the os.
- *Grade 2:* Placenta reaches the os (marginal).
- *Grade 3:* Placenta covers os but is positioned to one side (partial).
- *Grade 4:* Placenta is positioned squarely over the os (complete).

It can be diagnosed during routine antenatal ultrasound screening and is classically associated with painless vaginal bleeding. Placenta previa can occur in up to 10% full-term pregnancies. If placenta previa is suspected then avoid PV examination as it can result in massive blood loss.

Risk factors:

- Previous cesarean section
- Multiple pregnancy
- Multipara
- Previous myomectomy.

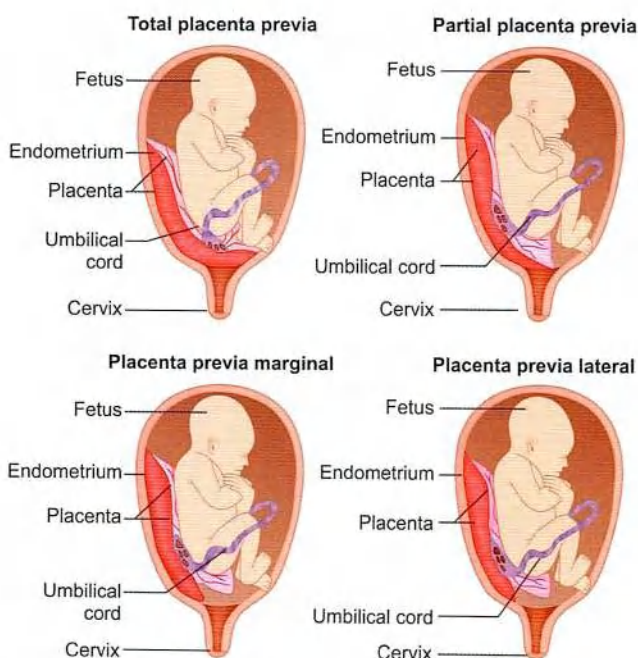


Fig. 1: Types of placenta previa.

Placental Abruption

It is the premature separation of a normally implanted placenta with bleeding that can be either concealed or per vagina. It usually presents with painful vaginal bleeding and is associated with uterine tenderness. Much of the bleeding is concealed. It can be diagnosed with ultrasonography (USG) or magnetic resonance imaging (MRI). It presents a significant risk to both mother and fetus.

Risk factors:

- Maternal hypertension
- Uterine over distention
- Previous abruption
- Abdominal trauma
- Smoking
- Postamniocentesis
- High parity.

Complications:

- Anemia
- Coagulopathy including disseminated intravascular coagulation (DIC)
- Acute renal failure
- Uterine atony
- Fetal distress—fetal death.

Uterine Rupture

It is an uncommon but potentially fatal to both mother and baby.

Risk factors:

- Previous uterine incision
- Use of prostaglandins to stimulate labor following a previous LSCS

- Uterine trauma
- Uterine anomalies
- Tumors
- Fetal malposition.

Rupture may be painless with the early signs being limited to slow progress in labor and deterioration in the cardiotocogram (CTG) (**Fig. 2**). A high degree of suspicion is required in a patient in labor after previous cesarean section. Hemorrhage can be torrential, accompanied by hypotension, abdominal pain and a change in uterine contour.

Treatment: A general anesthetic is the choice as there is rapid decompensation of the patient.

The management is directed at:

- Resuscitation of mother
- Emergency delivery of baby
- Repair of uterus or hysterectomy
- Preparation for the management of massive blood loss
- Invasive monitoring
- Intraoperative serial investigation—complete blood count, coagulation profile, electrolytes
- High dependency unit (HDU)/intensive care unit (ICU) care postoperatively.

Postpartum Hemorrhage

It can be primary PPH if blood loss occurs within 24 hours of delivery or secondary PPH if blood loss occurs over 24 hours following delivery up to 6 weeks postdelivery.

Postpartum hemorrhage can be classified as minor (with 500–1,000 mL blood loss), moderate (1,000–2,000 mL blood loss), or severe (>2,000 mL blood loss).

Incidence: 5–10% of deliveries.

Uterine Atony

It accounts for 80% of primary PPH.

Risk factors:

- Prolonged, augmented or precipitate labor
- Uterine over distention
- Placenta previa
- Increasing parity
- Advanced maternal age.

Treatment:

- Physical compression of uterus by bimanual compression or uterine massage.
- Drugs:
 - Intravenous infusion of oxytocin
 - Intramuscular (IM) ergometrine
 - Prostaglandins.

Trauma

Trauma to the perineum, cervix and vagina are common after normal vaginal delivery, the risk further increased by instrumentation (forceps delivery or vacuum extraction). In a well-contracted uterus with ongoing bleeding, genital tract trauma must be ruled out.

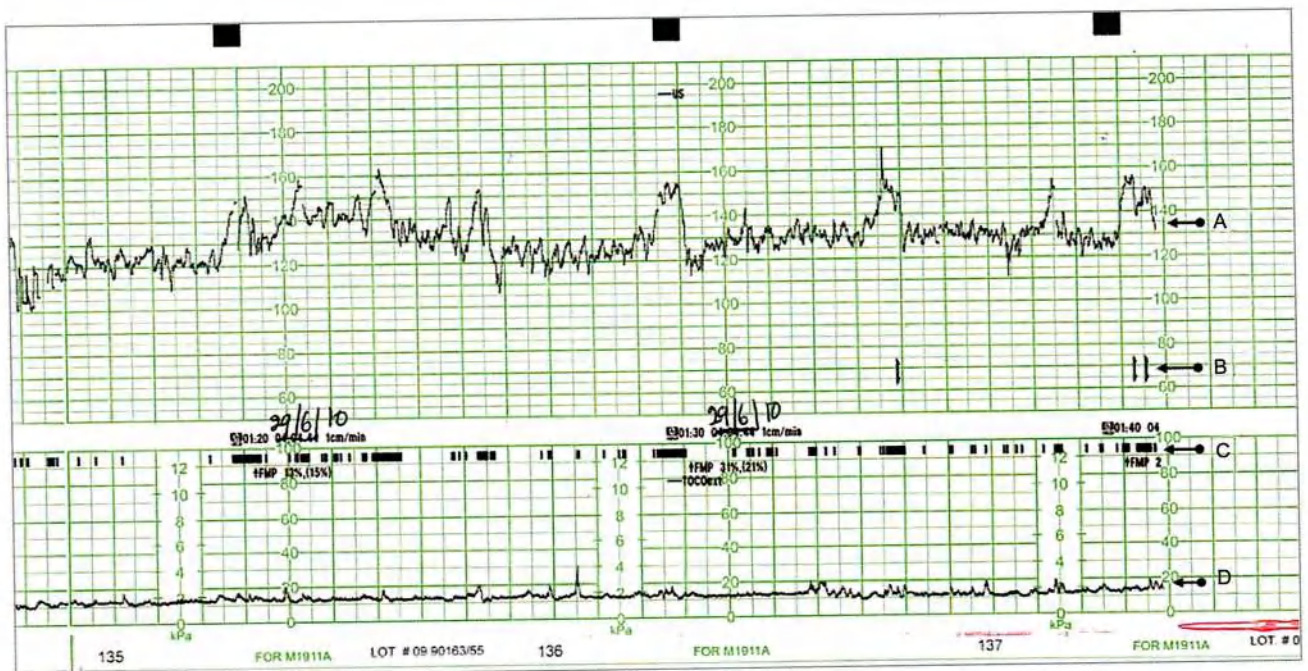


Fig. 2: Cardiotocograph of a woman not in labor. [A: fetal heartbeat; B: indicator showing movements felt by mother (caused by pressing a button); C: fetal movement; D: uterine contractions].

Retained Placenta

It is another common cause of both primary and secondary PPH.

Uterine Inversion

Uterine inversion is a rare but serious condition. Early reduction of the uterus is vital and this may require uterine relaxation; nitrates—50 µg bolus, β₂-agonists and magnesium have all been successfully used.

Risk factors:

- Uterine atony
- Uterine anomalies
- Inappropriate fundal pressure
- Excessive umbilical cord traction.

Coagulopathy

Both congenital and acquired, contribute to massive obstetric hemorrhage.

Q. What are the complications associated with APH?

Ans.

Fetal complications:

- Premature delivery
- Growth retardation
- Intrauterine death
- Congenital malformations
- Birth asphyxia.

Maternal complications:

- Acute hemorrhagic shock
- Acute kidney injury—renal tubular necrosis
- Premature labor
- Postpartum hemorrhage
- Retained placenta
- Complications associated with massive blood transfusion
- Maternal death.

Q. What would be your management plan for anticipated massive obstetric hemorrhage such as a lady posted for cesarean section with a low-lying anterior placenta and previous uterine scar?

Ans. Pregnant women are often young, healthy and have an increased blood volume of up to 20% at term and therefore are likely to compensate well in case of hemorrhage until the circulating blood volume is very low.

Goals of Management

- Rapid access to circulation and infusion of fluid in the first instance with rapid availability and administration of blood
- Avoid complications of massive blood transfusion, i.e., acid-base disturbance, transfusion-related acute lung injury (TRALI), hypocalcemia, hyperkalemia, hypothermia, and thrombocytopenia

- Efficient teamwork involving senior members including anesthetist and obstetrician.

Management Plan in Case of Massive Obstetric Hemorrhage

- Two large-bore IV cannulas
- Rapid infusion device or pressure bags in theater
- Blood warmer and warming blanket
- Blood cross-matched and available
- Institute preoperative invasive monitoring
- Consider cell salvage if available
- Consider interventional radiological procedures, if available
- Repeated quantitative assessment of blood loss
- *Vasopressors:* After adequate volume replacement or to maintain MAP during ongoing hemorrhage, a phenylephrine infusion (25–50 µg/min) appears to be more effective than phenylephrine boluses to prevent hypotension.

Communication and teamwork are essential in cases of both anticipated and unanticipated maternal hemorrhage:

- Alert blood transfusion service and the hematologist
- Allocate roles to team members
- Ensure departmental guidelines exist for the management of massive obstetric hemorrhage.

Q. Enumerate the causes of DIC related to obstetrics.

Ans.

- Intrauterine death
- Amniotic fluid embolus
- Sepsis
- Preeclampsia
- Placental abruption
- Retained products of conception
- Induced abortion
- Excessive bleeding
- Acute fatty liver.

Q. What are the various treatment options for massive obstetric hemorrhage?

Ans.

Pharmacological

- Refer below.

Surgical Options

- Bimanual uterine compression
- B-Lynch suture (brace suture) is a continuous suture used to encircle and mechanically compress the uterus, with the aim of avoiding a hysterectomy
- Uterine tamponade by using a Rusch urological balloon or Sengstaken-Blakemore tube
- Compression/clamping aorta as a temporary measure to minimize blood loss while preparing for a permanent option

- Ligation of uterine and internal iliac arteries—can be technically difficult, and the presence of collateral vessels may reduce effectiveness
- Hysterectomy
- Uterine replacement in case of a uterine inversion.

Interventional Radiological Options

- Bilateral iliac artery balloons may be placed electively and inflated at cesarean section or should bleeding occur
- Selective uterine artery embolization.

Blood and Blood Products

- Transfusion of red blood cells should occur early and group-specific blood or O-negative should be used, until cross-matched blood is available
- Avoid dilutional coagulopathy with excessive crystalloid or colloid administration.

Regular measurements of complete blood count, fibrinogen levels and clotting are recommended to guide transfusion requirements.

Point-of-care testing: Appropriate administration of red cells, blood components and coagulation factors may be guided by of hemoglobin values, e.g., HemoCue, and TEG or ROTEM.

Fresh frozen plasma: It contains all coagulation factors. If prothrombin time/activated partial thromboplastin time is >1.5 times normal and hemorrhage is continuing.

Dose: 12–15 mL/kg.

Fibrinogen:

Aim: Fibrinogen level of >2 g/L should be maintained. Transfuse cryoprecipitate

Platelets: In major hemorrhage, aim to keep platelets $>50 \times 10^9/L$; platelets should be requested if there is ongoing bleeding and the platelet count has fallen below $100 \times 10^9/L$.

Q. Is intraoperative cell salvage (IOCS) an option in this scenario?

Ans. There is increasing evidence that cell salvage in obstetrics is safe unlike as previously thought of since the likely risks of amniotic fluid embolism, contamination with fetal debris and isoimmunization of a rhesus-negative mother by fetal red cells have not been proven nor is there any evidence to suggest the same. However, a separate suction should be used for the amniotic fluid to reduce contamination risk and an appropriate leukocyte depletion filter used to administer the salvaged blood. IOCS has been endorsed by NICE guidelines.

Q. What are placenta accreta, increta and percreta?

Ans. Placenta accreta is an abnormal attachment of the placenta to the uterine wall. Three grades have been identified, based on histopathologic assessment of myometrial invasion by the chorionic villi (**Fig. 3**):

- *Placenta accreta (75–78%):* It is when the chorionic villi attaches strongly to the myometrium but does not penetrate it. It is not restricted within the decidua basalis.

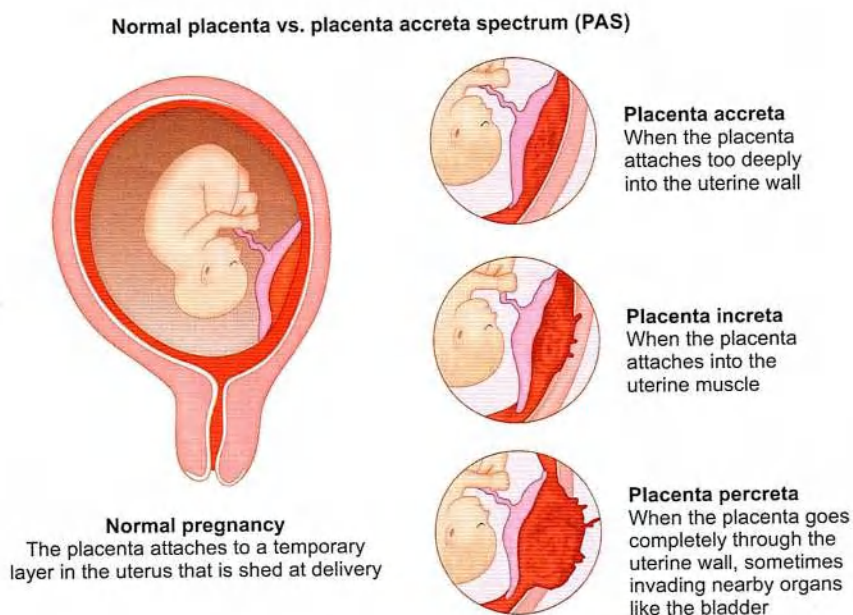


Fig. 3: Grades of placenta accreta and normal placenta.

- *Placenta increta* (17%): Chorionic villi invade into the myometrium.
- *Placenta percreta* (5–7%): It is the highest risk form of the condition, when the placenta chorionic villi penetrate the entire myometrium to the uterine serosa (through entire uterine wall). Here the placenta may invade the other organs such as the rectum or urinary bladder.

Risk factors:

- Prior cesarean section
- Uterine instrumentation or surgery
- Placenta previa.

Diagnosis:

- It is made by using transvaginal USG.

Complications:

- Prematurity
- Hemorrhage
- Transfusion related
- Increased incidence of hysterectomy.

Management of placenta accreta:

- *Conservative management:* Curettage and/or oversewing of the placental bed and occluding the blood vessels that supply the placenta.
- For majority of cases, hysterectomy remains the procedure of choice.

Q. A 24-year-old lady has a retained placenta. What precautions would you take while anesthetizing this lady?

Ans.

- If the placenta is not delivered within 30–60 minutes following the birth, it is considered a retained placenta.
- The retroplacental myometrium contracts following delivery of the baby which leads the placenta to shear away from its bed and be expelled. Retained placenta complicates 2% of deliveries worldwide.

Risk factors:

- Previous retained placenta
- Previous injury to uterus
- Preterm delivery
- Induced labor
- Multipara.

Potential complications:

- Primary PPH
- Secondary (delayed) PPH—due to retained placental tissue
- Uterine inversion
- Puerperal sepsis.

Active versus expectant management of the third stage of labor:

- *Active management* involves the administration of oxytocin after delivery followed by early clamping and

cutting of the umbilical cord. Controlled cord traction is applied with simultaneous suprapubic pressure to prevent inversion of the uterus.

- *Expectant* (also known as conservative or physiological) *management* involves waiting for signs of spontaneous separation and delivery of the placenta.

Signs of placental separation:

- Uterus rises in maternal abdomen
- Uterine shape changes from discoid to globular
- Umbilical cord lengthens
- Vaginal blood loss.

Management: Observe the patient for signs of blood loss, e.g., pallor, tachycardia, and hypotension (may be delayed). Blood loss may be concealed and difficult to estimate. Choice of anesthesia will depend on severity of bleeding and hemodynamic stability of the patient:

- Two large-bore IV cannulas
- Fluid resuscitation with crystalloid/colloid
- Blood cross-matched and available
- Aspiration prophylaxis.

Anesthetic technique: Refer **Table 1**.

TABLE 1: Comparison of general anesthesia (GA), regional anesthesia and sedation.

Technique	Advantages	Disadvantages
General anesthesia	Dose-dependent uterine relaxation by volatile agents	Risks of GA, e.g., airway compromise, failed intubation, aspiration, anaphylaxis
Spinal	• Rapid establishment of profound analgesia • Avoids risks of GA	Potential for sudden hypotension if extent of hemorrhage is underestimated
Epidural	Good if already in situ	Takes time to establish one in case of emergency as well as to achieve surgical anesthesia
Sedation	Quick and easy	• Poor uterine relaxation • Unprotected airway: risk of aspiration if overdose

Note: Intravenous nitroglycerin (NTG) 50–100 µg has been used effectively to relax the uterus under close hemodynamic monitoring.

General anesthesia: Adequate preoxygenation followed by rapid sequence induction with an endotracheal intubation is the gold standard. The choice of induction agent would vary according to the hemodynamic stability of the patient. In this scenario, IV induction agents like etomidate and ketamine should be considered. Volatile agents depress uterine contractility in a dose-dependent manner.

Regional anesthesia: Spinal anesthesia eliminates the risks associated with general anesthesia. Approximately 1.5–2.0 mL of hyperbaric 0.5% bupivacaine would be adequate to attain the desirable level of surgical anesthesia and maternal intraoperative comfort. A low-dose spinal anesthetic regimen comprising, 1.2 mL of 0.5% bupivacaine and 25 µg fentanyl can also be considered as another option.

Hypotension secondary to regional anesthesia is likely to be related to maternal blood loss rather than block height.

Q. What drugs are useful (main or as adjuncts) in the management of obstetric hemorrhage (Table 2)?

Ans.

Oxytocin

A synthetically produced hormone, causes uterine contraction. It acts on oxytocin-specific receptors in uterine myometrium promoting uterine smooth muscle contraction. The dose is 5–10 IU bolus followed by an infusion at 5–10 IU/h used in:

- Induction and acceleration of labor
- Missed and incomplete abortion
- Postpartum hemorrhage (atonic uterus).

Side effects:

- Hypotension and reflex tachycardia due to peripheral vasodilatation
- Antidiuretic properties in high doses
- May prolong the QT interval and cause T-wave flattening.

Ergometrine

An ergot alkaloid derivative that stimulates contractions of uterine and vascular smooth muscle. Like other ergot alkaloids, ergometrine produces arterial vasoconstriction by stimulation of alpha-adrenergic and serotonin receptors and inhibition of endothelial-derived relaxation factor release.

Dose: 0.5 mg IM or 0.1 mg slow IV injection over 10 minutes.

Action: It produces effective uterine constriction.

Side effects:

- Severe nausea and vomiting
- Systemic vasoconstriction can produce hypertension
- Diarrhea
- Dizziness
- Hallucinations
- Vertigo and tinnitus.

Contraindications: Ergometrine is contraindicated in patients with:

- Eclampsia, preeclampsia or a history of hypertension
- Peripheral vascular disease or heart disease
- If there is any suspicion of retained placenta.

Carboprost

Carboprost (Hemabate) is a prostaglandin F₂-alpha (PGF₂α) and potentiates the uterotonic effect of oxytocin.

It is used to treat PPH due to uterine atony in patients unresponsive to ergometrine and oxytocin.

Dose: 0.25 mg into the myometrium or IM every 10–15 minutes till a maximum of 2 mg.

Action: Uterine constriction.

Caution:

- It can lead to severe nausea and vomiting
- May cause bronchospasm (avoid in asthmatics)
- Excessive dosage may cause uterine rupture.

Contraindications:

- History of glaucoma or raised intraocular pressure
- Uterine scars.

Misoprostol

Misoprostol is a synthetic prostaglandin E₁ (PGE₁) analog. It is used to treat PPH due to uterine atony in patients unresponsive to ergometrine and oxytocin.

Dose: 400–800 µg (2–4 × 200 µg tablets). The sublingual, oral, rectal and intrauterine routes have all been used successfully.

Side effects: Nausea, vomiting, diarrhea, abdominal pain, dyspepsia, flatulence, rashes and dizziness.

TABLE 2: WHO recommendations for uterotonics for PPH prevention.

Context	Recommendation
Efficacy and safety of uterotonics for PPH prevention	1. The use of an effective uterotonic for the prevention of PPH during the 3rd stage of labor is recommended for all births. To effectively prevent PPH only one of the following uterotonics should be used: – Oxytocin (recommendation 1.1) – Carbetocin (recommendation 1.2) – Misoprostol (recommendation 1.3) – Ergometrine/methylethergometrine (recommendation 1.4) – Oxytocin and ergometrine fixed dose combination (recommendation 1.5)
	1.1. The use of oxytocin (10 IU, IM/IV) is recommended for prevention of PPH for all births
	1.2. The use of carbetocin (100 µg, IM/IV) is recommended for prevention of PPH for all births in contexts where its cost is comparable to other effective uterotonics
	1.3. The use of misoprostol (either 400 µg or 600 µg PO) is recommended for the prevention of PPH for all births
	1.4. The use of ergometrine/methylethergometrine (200 µg, IM/IV) is recommended for the prevention of PPH in contexts where hypertensive disorders can be safely excluded prior to its use
	1.5. The use of oxytocin and ergometrine fixed-dose combination (5 IU/500 µg, IM) is recommended for the prevention of PPH in contexts where hypertensive disorders can be safely excluded prior to its use

Additional Drugs*Recombinant Factor VIIa (NovoSeven)*

It is not currently licensed for use in obstetrics. The use of rFVIIa is limited to patients with refractory hemorrhage; where embolization is unavailable; or hysterectomy is the only alternative. Recombinant factor VIIa is an expensive and controversial therapy with a chance of occurrence of a thromboembolic event. It causes a thrombin burst, promoting clotting in open vessels.

Dose: 90–100 µg/kg.

Disadvantage: Increases incidence of thrombotic complications.

Antifibrinolytics—Tranexamic Acid

Tranexamic acid is a potent antifibrinolytic agent that exerts its effects by blocking lysine binding sites on plasminogen molecules and has the potential to enhance the effectiveness of the patient's own hemostatic mechanisms. Consequently, clot breakdown (fibrinolysis) is inhibited and bleeding is reduced. The use of tranexamic acid should be considered in addition to oxytocin, at cesarean section to reduce blood loss in women who are at increased risk of PPH.

Dose: The initial dose is slow IV bolus of 1 g followed by a further 1 g 4 hours later.

Side effects: Potential risk of thrombosis. It may cause acute renal failure due to thrombosis-induced cortical necrosis.

Intravenous Calcium

Correcting calcium levels, especially in cases of massive transfusion, in order to optimize coagulation as well as uterine muscle contraction.

SUGGESTED READING

1. Allman KG, Wilson IH. Oxford Handbook of Anaesthesia, 4th edition. United Kingdom: Oxford University Press; 2016.
2. B-Lynch C, Coker A, Lawal AH, Abu J, Cowen MJ. The B-Lynch surgical technique for the control of massive postpartum hemorrhage: an alternative to hysterectomy? Five cases reported. *Br J Obstet Gynaecol.* 1997;104:372-5.
3. Muñoz M, Stensballe J, Ducloy-Bouthors AS, Bonnet MP, De Robertis E, Fornet I, et al. Patient blood management in obstetrics: prevention and treatment of postpartum haemorrhage. A NATA consensus statement. *Blood Transfus.* 2019;17(2):112-36.
4. National Institute for Health and Clinical Excellence. Intraoperative blood cell salvage in obstetrics. NICE Interventional Procedure Guidance No. 144. 2005.
5. Sentilhes L, Lasocki S, Ducloy-Bouthors AS, Deruelle P, Dreyfus M, Perrotin F, et al. Tranexamic acid for the prevention and treatment of postpartum haemorrhage. *Br J Anaesth.* 2015;114:576-87.
6. Solomon C, Collis RE, Collins PW. Haemostatic monitoring during postpartum haemorrhage and implications for management. *Br J Anaesth.* 2012;109:851-63.
7. Thomas D, Wee M, Clyburn P, Walker I, Brohi K, Collins P, et al. Blood transfusion and the anaesthetist: management of massive haemorrhage. *Anaesthesia.* 2010;65(11):1153-61.
8. Trikha A, Singh PM. Management of major obstetric haemorrhage. *Indian J Anaesth.* 2018;62(9):698-703.
9. Vogel JP, Williams M, Gallos I, Althabe F, Oladapo OT. WHO recommendations on uterotonics for postpartum haemorrhage prevention: what works, and which one? *BMJ Glob Health.* 2019;4(2):e001466.
10. World Health Organization. WHO recommendations for the prevention and treatment of postpartum haemorrhage. Geneva: WHO; 2012.

Hydrocephalus

Sheila Nainan Myatra, Sumitra Ganesh Bakshi, Shilpushp Jagannath Bhosale

A 3-year-old male (14 kg) child presents with history of:

- Frequent headaches and blurring of vision — Since 2 months
- Altered mental status — Since 3 days
- Vomiting 8–10 episodes — Since 2 days
- Fever 102°F — Since 1 day

Computed tomography (CT) scan reveals an obstruction within the ventricular system and he is planned for ventriculoperitoneal shunt procedure.

Q. What is hydrocephalus? What is the incidence of this condition?

Ans. Hydrocephalus, one of the most common pediatric and adult neurosurgical disorders, is an abnormal increase in the amount of cerebrospinal fluid (CSF) resulting from a disturbance of formation, flow or absorption of CSF thus resulting in enlarged cerebral ventricles. This may be congenital or acquired. Patients with hydrocephalus usually present with an increase of intracranial pressure (ICP).

Hydrocephalus can occur at any age depending on the etiology. Incidence of congenital hydrocephalus is 3/1,000 live births, whereas the incidence of acquired hydrocephalus is not known exactly.

Q. Where is CSF produced and how does it flow? What is the normal CSF pressure and what are the common causes of CSF blockade?

Ans. Cerebrospinal fluid is produced by the choroid plexus of the ventricles and circulates in one direction from the lateral ventricles to the 3rd ventricle and through the aqueduct of Sylvius to the 4th ventricle. From the 4th ventricle, CSF exists the brain through three separate openings, one in the midline (foramen of Magendie) and one on either side (foramina of Luschka). It enters the subarachnoid space at the foramen magnum, circulation down the spine, and then circulates up again to the surface of the brain where it is absorbed at the arachnoid granulations. These are sieve-like structure where the CSF enters the venous circulation, leading to the sagittal sinus.

The rate of CSF absorption is driven by the ICP, while arachnoid granulations and the intracranial venous sinus pressure provide the resistance to CSF outflow. The pressure within the ventricles is driven by Laplace's law suggesting that the pressure required to overcome the tension in the surrounding cerebral mantle is less in enlarged ventricles than it is in smaller ventricles explaining the CSF flow in enlarged ventricles in chronic hydrocephalus.

In the adult human, CSF is formed at a rate of about 0.5 mL/min. The total volume is about 200 mL, of which 30 mL is in the ventricles and the remainder in the subarachnoid space. The circulation of CSF leads to the fluid being completely replaced about every 4 hours.

Normal CSF pressure when one is lying down is about 115 cm of H₂O with a range of 70–180 cm H₂O. In vertical position, the CSF pressure is higher (375–550 cm H₂O).

Common Causes of CSF Fluid Blockade

- *Infectious:* Meningitis, encephalitis, abscess
- *Neoplastic:* Astrocytoma, meningioma, choroid plexus papilloma, medulloblastoma
- *Vascular:* Arteriovenous malformations, aneurysm
- *Congenital:* Neural tube defects, genetic defects, e.g., Chiari malformation, arachnoid cysts, colloid cysts, encephalocele.

Q. Give the etiological classification of hydrocephalus. What is the pathophysiology of this condition?

Ans.

- Overproduction of CSF—choroid plexus papilloma
- Blockage of the normal flow of CSF:

- *Communicating or absorptive*—blockage of the resorption of CSF in the arachnoid villi, basal cisterns, or subarachnoid space. The ventricles are patent and all four are enlarged:
 - Inflammatory—infectious ventriculitis, chemical ventriculitis, intraventricular hemorrhage. In these cases, the arachnoid granulations are occluded by protein or blood degradation products rendering CSF absorption relatively ineffective.
 - Impaired CSF absorption in spina bifida.
- *Noncommunicating*: Obstruction proximal to the foramina of Luschka and Magendie at the outlet of the 4th ventricle:
 - Cysts
 - Tumors—tumors of the posterior cranial fossa
 - Infection and hemorrhage
 - Congenital malformations—aqueduct stenosis, Arnold–Chiari malformation, Dandy–Walker cyst
 - Aqueductal stenosis.

The classical distinction between obstructive and communicating hydrocephalus is less useful clinically as the cause of reduced absorption of CSF in communicating hydrocephalus is usually functional obstruction at the arachnoid villi (e.g., by blood or protein).

Due to the above factors, CSF accumulation leads to ventricular enlargement and rises in intraventricular (and hence intracranial) pressure. In infants with open fontanelles, some of this rise in pressure is counteracted with enlargement of the head. When the maximum capacity for head enlargement has been used, rapid deterioration follows because of raised ICP.

In neonates, hydrocephalus may exist despite normal ICP an increase in CSF volume is compensated by a decrease in brain mass until the compliance limit of immature brain tissue is reached.

Older children with closed fontanels develop clinical signs of intracranial hypertension without progressive head enlargement.

Q. Enumerate some of the common syndromes and anomalies associated with this condition.

Ans.

Meningomyelocele: Myelomeningocele is protrusion of the meninges through a midline bony defect of the spine, forming a sac containing CSF and neural tissue.

Aqueduct stenosis: In newborn children, common cause of hydrocephalus is obstruction of the aqueduct of Sylvius. Obstruction here prevents the free passage of CSF from the lateral and 3rd ventricles to the 4th ventricle, and from thereon to the subarachnoid space.

Arnold–Chiari syndrome: Malformation of the brain. It consists of a downward displacement of the cerebellar tonsils and the medulla through the foramen magnum. Symptoms include cerebellar symptoms, headache, visual disturbances, nausea, extreme muscle soreness, facial pain, hearing problems, insomnia, pain in the neck/upper arm, tingling, burning. Other conditions, sometimes associated with Chiari malformation include hydrocephalus, spina bifida, and connective tissue disorders such as Ehlers–Danlos syndrome and Marfan’s syndrome.

Dandy–Walker syndrome: Dandy–Walker complex is a congenital brain malformation involving the cerebellum and the fluid-filled spaces around it. Symptoms of increased ICP and signs of cerebellar dysfunction such as unsteadiness, lack of muscle coordination or jerky movements of the eyes may occur. Other symptoms include increased head circumference, bulging at the back of the skull, problems with the nerves that control the eyes, face and neck, and abnormal breathing patterns.

Intracranial Tumor

Mucopolysaccharidoses which obliterate the subarachnoid space—group of metabolic disorders caused by the absence or malfunctioning of lysosomal enzymes needed to break down molecules called glycosaminoglycans.

Achondroplastic disorders (which alter occipital bone growth and impede venous outflow):

- Arachnoid cyst
- Porencephaly
- X-linked hydrocephalus
- In utero intraventricular hemorrhage
- Maroteaux–Lamy syndrome.

Q. What is the clinical presentation of hydrocephalus in an infant and in an older child?

Ans. The clinical features of hydrocephalus depend on the age of the patient, the pathology and site of CSF obstruction. Acute-onset cases usually present with symptoms of raised ICP and the ventricles may not be markedly enlarged. In chronic hydrocephalus, the ventricles are usually enlarged.

Infant and Newborn

- Cranium enlarges at rate > facial growth
- Fontanelle full and bulging
- Poor head control
- Irritability, lethargy
- Delayed milestones
- Nausea/vomiting
- Enlargement and engorgement of the scalp veins—due to reversal of flow from the intracerebral sinuses due to increased ICP
- Macewen’s sign cracked pot sound on percussing overdilated ventricles

- *Setting sun sign (upward gaze palsy)*: Parinaud's syndrome from pressure on the region of suprapineal recess
- Hyperactive reflexes
- Cranial nerve palsies:
 - Lateral gaze palsy (6th nerve—abducens palsy): The long intracranial course is postulated to render this nerve sensitive to pressure (more common with Arnold–Chiari malformation)
 - Stridor due to bilateral vocal cord palsy due to stretching of the vagus nerve over the lip of the jugular foramen
 - Cortical blindness (optic nerve)
 - Visual backing disorder (oculomotor nerve)
- Irregular respiration and apneic spells
- Splaying of cranial sutures (seen on plain X-ray skull).

Older Child/Adults with Rigid Cranial Vault

Symptoms of increased ICP including papilledema, headache, nausea, vomiting, gait changes, upward gaze, and abducens palsy. Slowly enhancing ventricles may initially be asymptomatic.

Chronic hydrocephalus:

- Beaten copper cranium (beaten silver appearance) on plain X-ray
- Third ventricle herniating into the sella [on computed tomography (CT), magnetic resonance imaging (MRI)]
- Erosion of sella turcica
- Macrocrania
- Atrophy of corpus callosum
- In infants—sutural diastasis, delayed closure of fontanelles, developmental delay and failure to thrive.

Signs and symptoms of hydrocephalus are due to the acute or chronic rise in ICP. Acute hydrocephalus may be caused by intraventricular hemorrhage in the premature infant, hemorrhage into a tumor, or expansion of a colloid cyst of the 3rd ventricle. The symptoms may be unrecognized in nonverbal and developmentally delayed child. Vomiting, dehydration, lethargy, neurogenic pulmonary edema, and coma are indications of emergency decompression to prevent herniation of the brainstem, respiratory and cardiac arrest and possibly death.

Chronic hydrocephalus can be due to congenital aqueductal stenosis, meningitis, and spinal cord tumors.

Slowly progressive signs like irritable behavior, poor school performance, intermittent headaches, rambling speech, altered behavior, confusion lethargy, unsteady gait, seizures, incontinence and papilledema may be present.

In neonates, nonfusion of the cranial sutures causes widening of the sutures and enlargement of the head circumference causing airway obstruction.

In infants and small children, vomiting and decreased oral intake are common presentations and can be misdiagnosed as viral illness or gastroenteritis. Acute obstructive hydrocephalus in undiagnosed intracranial neoplasm may lead to sudden and unexpected death due to cerebral herniation.

Q. What is normal pressure hydrocephalus (NPH) and what are its characteristics?

Ans. Normal pressure hydrocephalus is a chronic type of communicating hydrocephalus whereby the increase in ICP due to accumulation of CSF becomes stable and that the formation of CSF equilibrates with absorption. The ICP gradually falls but still maintains a slightly elevated level and the CSF pressure reaches a high normal level of 150–200 mm H₂O. Because of this equilibration, patients do not exhibit the classic signs of increased ICP such as headache, nausea, vomiting, or altered consciousness. However, patients do exhibit the classic Hakim triad of symptoms includes gait difficulties, urinary incontinence, and mental decline (dementia) as first described by Hakim and Adams in 1965. It is often misdiagnosed as Parkinson's disease, Alzheimer's disease, and senility due to its chronic nature and its presenting symptoms.

Although the exact mechanism is unknown, NPH is thought to be a form of communicating hydrocephalus with impaired CSF reabsorption at the arachnoid villi.

Q. How will you diagnose this condition?

Ans.

- History and clinical findings
- Serial head circumference measurements
- Skull radiographs
- CT scan confirms diagnosis
- Shunt scan (in patients with a shunt to determine the site of malformation)
- Fundus examination
- *Venous ophthalmodynamometry*: This technique may be used in the differential diagnosis of malfunction of ventricular shunts, gastrointestinal disorders, hypertensive hydrocephalus, and brain atrophy
- *Transcranial Doppler*: It is a noninvasive method of evaluating hydrocephalus. Decrease in diastolic velocity and increase in pulsatility index (systolic velocity – diastolic velocity/mean velocity) are diagnostic.

Q. What is the medical treatment of this condition?

Ans. Hydrocephalus remains a surgically treated condition. *Diuretic therapy*: It may be tried in premature infants with bloody CSF (as long as there is no evidence of active hydrocephalus) while waiting to see if there is resumption of normal CSF absorption. However, this is considered as an adjunct to definitive treatment or as a temporizing measure.

Satisfactory control has been seen in 50% of children < 1 year who had stable vital signs and no symptoms of elevated ICP using the following:

- *Acetazolamide (a carbonic anhydrase inhibitor)*: 25 mg/kg/day PO divided TID increase by 25 mg/kg/day until 100 mg/kg/day is reached
- *Simultaneously start furosemide*: 1 mg/kg/day PO divided TID
- Osmotic diuretic—not preferred because of rebound phenomenon
- Correct acidosis
- Watch for and correct electrolyte abnormalities
- Watch for acetazolamide side effects—lethargy, tachypnea, diarrhea
- Perform weekly ultrasound or CT scan and insert ventricular shunt if ventriculomegaly occurs.

Spinal taps: Hydrocephalus after intraventricular hemorrhage may be only transient. Serial taps [ventricular or lumbar puncture (LP)] may temporize until resorption resumes, but LPs can only be performed in communicating hydrocephalus. If spontaneous resorption does not occur, shunts will be required.

Q. Describe in detail the common neurosurgical procedures for this condition.

Ans. An emergency ventriculostomy or extracranial ventricular drainage may be performed before the direct surgical introduction of a ventricular shunt.

For ventriculostomy, the right frontal region (frontal horn of the lateral ventricle) is preferred because it is rarely the dominant hemisphere and this can be done at the bedside in intensive care unit (ICU). The other end of the extraventricular drain (EVD) is connected to an extension tube and the drainage burette that empties into a collecting bag. Ventriculostomy also allows for monitoring of ICP in the postoperative period where the burette is zeroed at the level of foramen of Monro which corresponds to the external auditory meatus in the supine position. Complications with EVD include infection, hemorrhage, epileptic seizures, displacement of catheter, etc.

Choroid plexectomy: For communicating hydrocephalus, open surgery is associated with a higher mortality.

Eliminating the obstruction (e.g. opening the stenosed Sylvian aqueduct): Often has higher morbidity and lower success rate than simple CSF diversion with shunts, except perhaps in case of a tumor.

Third ventriculostomy: There is a resurgence of interest in this procedure due to the increased ventriculoscopic surgery. Developments in optics, image processing and improvements in anesthesia have enabled endoscopic

third ventriculostomy to be used safely with comparable results to ventriculoperitoneal (VP) shunts. It is usually done in patients with obstructive hydrocephalus. It may be an option for managing shunt infection as a means to remove hardware without subjecting the patient to raised ICP. This has also been proposed as an option for patients who develop subdural hematomas after shunting.

Shunting: Different types of shunt procedures can be performed:

- Ventriculoperitoneal shunt
- Ventriculoatrial (VA) shunt
- Ventriculopleural shunt
- Torkildsen shunt
- Lumboperitoneal shunt.

Q. Describe the ventricular shunts available for this condition.

Ans. In most cases, surgical treatment of hydrocephalus consists of insertion of a ventricular shunt. The shunt is an artificial device designed to maintain normal ICP.

Shunts usually consist of three parts:

1. Proximal end is radiopaque and is placed into the ventricle. This end has multiple small perforations.
2. Valve allows for unidirectional flow. It can adjust various opening pressures. Usually has a reservoir that allows for checking shunt pressure and sampling CSF.
3. Distal end is placed into the peritoneum or another absorptive surface by tracking the tubing subcutaneously.

Essentially two types of shunts are available: (1) the pressure-regulating shunt and (2) the flow-regulating shunt. The pressure-regulating shunts are designed to maintain a difference of pressure between their inlet and outlet, and they allow flow of CSF once that preset pressure has been reached. The flow-regulating shunts are designed to allow a constant flow of CSF, simulating the normal flow of CSF. The differential pressure valves are more prone to cause overdrainage complications, whereas the flow-regulating valves are more prone to valve obstruction. To avoid overdrainage, the differential pressure valves are further divided into low pressure (20–40 mm H₂O), medium pressure (40–70 mm H₂O), and high pressure (80–100 mm H₂O).

Shunt insertion is done through parieto-occipital burr hole on the nondominant side so as to place the ventricular catheter in the frontal horn of the lateral ventricle. With this approach, the shunt valve is situated behind the ear and is easily palpable by the parents and patients through the skin. The distal catheter then drains the CSF from one-way valve to one of the extracranial locations in the body from where it gets absorbed.

The VP shunt is small tubing that is placed inside the brain's ventricle and tunneled underneath the skin to

the peritoneum. The purpose of the VP shunt is to reduce the amount of CSF in the brain by draining it into the abdominal (peritoneal) space. The peritoneum is the usual, safe site and also the most preferred to place the end of the ventricular shunt.

Certain circumstances may require placing the end of the shunt tubing into a blood vessel leading to the heart (ventriculovascular or VA) or into the pleural space of the chest (ventriculopleural). In VA shunt, the distal tube is put under the fluoroscopic guidance. CT scan is done before discharge to verify the position of the ventricular catheter and serves as future reference. Other options may be the gallbladder, ureter, and urinary bladder.

Q. What are the common complications of shunt procedures?

Ans. Common to all types of shunts:

- Malfunction—because of obstruction, disconnection, breakage, migration
- Infection
- Overdrainage—early overdrainage can cause subdural hematoma. Chronic overdrainage can lead to intermittent shunt obstruction due to slit ventricle symptom. It can also give rise to secondary craniosynostosis and thick skull (hyperostosis cranii ex vacuo)
- Pneumocephalus
- Seizures—5.5% risk of seizures in the first year
- Hardware erosion through skin
- Act as a conduit for extraneural metastasis of certain tumors
- Silicone allergy.

Complications unique to ventricular peritoneal shunts:

- About 17% incidence of inguinal hernia
- Obstruction of peritoneal catheter—due to occlusion by omentum, by peritoneal cyst, severe peritoneal adhesions
- Peritonitis from shunt infection
- Hydrocele
- CSF ascites
- Tip migration—into scrotum, through diaphragm, perforation of viscus
- Intestinal obstruction, volvulus
- Overshunting.

Complications unique to ventricular atrial shunts:

- Requires repeated lengthening in a growing child
- Higher risk of infection
- Possible retrograde flow of blood into the ventricles if valve malformation
- Shunt embolism
- Vascular complications—thrombophlebitis, pulmonary microemboli, pulmonary hypertension

- Shunt nephritis (immune complex deposition in kidneys).

Q. What mechanisms regulate cerebral blood flow (CBF)?

Ans. Cerebral perfusion pressure (CPP) is defined as the difference between the mean arterial pressure (MAP) and the sum of ICP and the central venous pressure (CVP):

$$CPP = MAP - (ICP + CVP)$$

The cranial contents consist of brain tissue (80%), blood (10%), and CSF (10%). The volume of the cranial vault is fixed once the sutures of the skull have become fused, normally by 2 years of age. In the neonate, even though the volume of the vault can expand if the increase in intracranial contents occurs gradually, such as in congenital hydrocephalus, this accommodation has a maximum limit, which once exceeded will lead to an increase in ICP. Any changes in the volume of the individual cranial contents can lead to an alteration in ICP and this has both pathological and therapeutic implications.

Cerebral blood flow determines cerebral blood volume (CBV) and is higher in children compared to adults (100 vs. 50 mL/100 g/min). From 6 months to 3 years, CBF is around 90 mL/100 g/min and from 3 to 12 years, it is around 100 mL/100 g/min. It is coupled to the metabolic demands of the normal brain and regulated via oxygen requirement [cerebral metabolic rate of oxygen (CMRO₂)], arterial partial pressure of carbon dioxide (PaCO₂) and intracerebral acidosis. In children, CMRO₂ is approximately 5.2 mL/100 g/min compared to 3.5 mL/100 g/min in adults. Autoregulation allows for a relatively constant blood flow across a wide range of arterial pressures, which can be as low as 40 mm Hg in small children. This mechanism is not available to premature neonates or during some pathological processes (e.g., infection) where the relationship between CBF and arterial pressure is more linear. Reducing PaCO₂ causes vasoconstriction of the cerebral vessels, reducing CBF, blood volume and ICP but at the expense of oxygen delivery. Hyperventilation should only be used as a short-term measure when ICP is high.

Cerebrospinal fluid is continuously produced by the choroid plexus, and after circulating through the ventricles is absorbed at the arachnoid villi. In children, the rate of production of CSF is 0.2–0.4 mL/min with around 250 mL produced and absorbed per day and at any given time around 70 mL is present in the head. Any interruption to normal flow, increased production or decreased reabsorption of CSF can manifest itself as hydrocephalus.

Q. What investigations will you ask for this child?

Ans.

- Complete blood count
- Erythrocyte sedimentation rate (ESR)

- Renal function
- Serum electrolytes
- CT scan
- Investigations related to coexisting medical condition or syndrome.

Q. What are the important points to be considered while taking preoperative history and examination?

Ans. The history and examination is an essential component of the preoperative assessment and should include:

- Standard anesthetic history
- Birth history (prematurity)
- Immunization history
- Family history
- History of any allergies
- History of problems with previous anesthesia/surgery
- Drug history: past medications, current medication (e.g., anticonvulsants, acetazolamide, furosemide)
- Fasting status
- Associated anomalies
- Possibility of comorbidities
- Systemic:
 - *Central nervous system:* Level of consciousness, cranial nerve palsies, signs of raised ICP, brainstem involvement (reduced level of consciousness and therefore increased risk of pulmonary aspiration).
 - *Respiratory system:* Assessment of the airway, bronchopulmonary dysplasia, kyphoscoliosis, recurrent respiratory infections secondary to neurological dysfunction. If the preoperative assessment reveals a patient with multiple comorbidities, such as premature infant with chronic lung disease, the need for additional respiratory support in the postoperative period should be considered and organized in advance.
 - *Cardiovascular system:* Congenital cardiac disease.
 - *Volume status* should be determined as prolonged vomiting and dehydration may necessitate intravenous (IV) fluid preoperatively.

Q. How will you optimize this patient prior to surgery?

Ans. This child will require immediate surgery in view of the clinical findings.

Antibiotics need to be started since the patient is febrile and shows clinical signs of infection.

Ensure adequate hydration and correction of any dyselectrolytemia if present in view of the persistent vomiting. Medical treatment may be started while awaiting surgery.

Q. What are the factors to be considered while premedicating the child for VP shunt?

Ans. Sedative premedication should be considered carefully as it may exacerbate or mask signs of neurological

dysfunction. Narcotics and sedatives may depress ventilation, resulting in further rise in ICP. As a general rule, preoperative sedation should not be used. However, an anxious combative patient may have a detrimental rise in ICP during induction. Hence, sedation should be given only if required in the above situation keeping in mind the factors stated. A titrated dose of oral or intranasal midazolam may be considered; however, the child should be carefully monitored while awaiting surgery for signs of neurological deterioration as this can occur rapidly.

Prophylactic antibiotics should be given to lower shunt infection rate and continued if already started in a child with signs of infection or if shunt revision is planned.

Q. What are the anesthetic considerations for ventriculo-peritoneal shunt?

Ans. The child should be carefully monitored while awaiting surgery for signs of neurological deterioration as this can occur rapidly. If patient suffers respiratory arrest or sudden deterioration in neurological status due to ICP and tonsillar herniation, endotracheal intubation and hyperventilation with 100% oxygen should be carried out and child should be prepared for urgent ventricular tap.

Latex precautions are recommended for patients with myelomeningocele undergoing shunt placement.

The method of induction is determined by the circumstances of the case and the preference of the patient and the anesthetist. The aim of anesthetic induction is to avoid increases in ICP associated with hypercapnia, hypoxia, and changes in MAP. Volatile agent may increase the CBF whereas hypotension will decrease the CPP.

Intravenous or inhalational induction can be used in a child with normal ICP. IV induction allows for rapid control of the airway in emergency situations (raised ICP) if the patient is not fasted and if no difficulties with the airway have been anticipated. This may be achieved using a suitable induction agent in a carefully titrated sleep dose after preoxygenation (propofol 2–4 mg/kg, thiopentone 3–5 mg/kg). Ketamine should not be used as this will increase the ICP.

Gaseous induction is an acceptable alternative with a nonirritant volatile anesthetic agent such as sevoflurane or halothane especially when IV cannula is not in place. However, IV induction should be preferred if child has signs of raised ICP, stupor or delayed gastric emptying.

Muscle relaxation can be achieved with the use of a neuromuscular blocking agent (NMBA).

If a rapid sequence induction is used, the small rise in ICP associated with the use of succinylcholine can be attenuated by the use of opioids or defasciculating dose of nondepolarizing NMBAs. Opioids such as fentanyl or remifentanyl can be used to attenuate the hypertensive responses to laryngoscopy, intubation, and surgery.

Endotracheal intubation: In case of a very large head, intubation can become technically difficult. Proper positioning using a pad or bolster below the shoulder can help overcome this problem. Lateral position may also be tried for intubation. Flexometallic tube may be used to avoid kinking during head rotation. The tube should be firmly secured.

Anesthesia can be maintained with a volatile agent and a mixture of oxygen and air. The aim is to maintain CPP until the raised ICP is relieved by positioning of the VP shunt. To do this hypotension should be avoided and minute ventilation controlled to maintain normocarbia to optimize CPP and avoid increases in ICP. Positive end-expiratory pressure (PEEP) should be minimized to avoid venous congestion in the head but may be used if there are difficulties in maintaining oxygenation.

Ventilation: Mild hypocapnia (PaCO_2 32–35 mm Hg) following tracheal intubation may prevent further elevation of PaCO_2 ; however, aggressively decreasing the PaCO_2 increases the risk of brain ischemia and is not currently recommended. Normocapnia should be maintained in patients with normal ICP.

Positioning during surgery: The head is turned away from the surgeon with a pad/bolster below the operative site. Extreme rotation of the head can impede venous return through the jugular veins and lead to impaired cerebral perfusion, ICP and cerebral venous bleeding. All anesthetic equipment and IV lines must be on the side of the anesthetist. The patient should be positioned to allow good access for the surgeon and to avoid any undue pressure on vulnerable areas.

Measures to maintain CPP and prevent rises in ICP.

Consideration should be given to optimizing venous drainage to increase CPP, reduce venous bleeding and improve the surgical field.

Temperature control: The core temperature of the patient should be monitored (rectal or esophageal) and warming devices (e.g., warm air blankets) used as required to maintain normothermia.

On cannulating the ventricle, blood pressure (BP) may drop abruptly as brainstem pressure is suddenly released.

The most stimulating parts of the surgery include the initial incision and tunneling under the skin. A short-acting opioid, such as fentanyl (1–3 $\mu\text{g}/\text{kg}$) or remifentanyl (1 $\mu\text{g}/\text{kg}$), or increased depth of anesthesia, can be used to attenuate the increase in heart rate and ICP.

Normal saline or Ringer's lactate can be used as maintenance fluid as they are mildly hyperosmolar and theoretically attenuates cerebral edema. 5% dextrose should not be used as this is hypoosmolar and can increase cerebral edema.

Prophylactic antiemetic should be given before 30–60 minutes of extubation.

At the end of the procedure, neuromuscular blockade can be antagonized using neostigmine combined with an anticholinergic (e.g., atropine). Most patients can be extubated once awake, avoiding hypercarbia, and with a technique which minimizes the risk of aspiration. Patients with severe neurological deficits may have poor airway control and can be more prone to postoperative respiratory problems.

Q. What is the relationship between CPP and ICP and what measures can we take to manipulate them in the intraoperative period?

Ans.

$$\text{CPP} = \text{MAP} - (\text{ICP} + \text{CVP})$$

Cerebral perfusion pressure can be increased by either increasing MAP (with fluids, vasoconstrictors or inotropes) or by reducing ICP (hyperventilation, mannitol, sedation and analgesia) and reducing CVP (head-up position, head midline, prevent obstruction of venous drainage of neck with by avoiding extreme neck rotation).

Most anesthetic agents cause a reduction in MAP but this potentially deleterious effect is usually offset by the corresponding reduction in the CMRO_2 .

Q. What are the indications for rapid sequence intubation in these children?

Ans. Children with a full stomach for emergency surgery, delayed gastric emptying, and vomiting are indications for rapid sequence intubation. Child with severe neurological compromise may have a gastrostomy tube preoperatively which should be aspirated before induction and left open to prevent gastric distention and regurgitation.

Q. How is the child positioned during surgery and in the postoperative period?

Ans.

During surgery: The head is turned away from the surgeon with a pad/bolster below the operative site. Extreme rotation of the head can impede venous return through the jugular veins and lead to impaired cerebral perfusion, raised ICP and cerebral venous bleeding. All anesthetic equipment and IV lines must be on the side of the anesthetist. The patient should be positioned to allow good access for the surgeon and to avoid any undue pressure on vulnerable areas.

Postoperatively: Patient should be nursed flat so as to avoid subdural hemorrhage which may occur because of rapid collapse of ventricles in head high position.

Q. Why is the use of nitrous oxide not recommended for this procedure?

Ans. The use of nitrous oxide is not recommended as it significantly increases the CBF and CBV which can

contribute to an elevation in ICP and also since it is associated with an emetic effect, it may confuse the evaluation of the patient postoperatively. There is also a potential risk of increase in postoperative pneumocephalus with use of nitrous oxide.

Q. Why is intraoperative spontaneous ventilation not recommended during this surgery?

Ans. Intraoperative spontaneous ventilation is not recommended due to the risk of air embolism during craniotomy and VA shunt placement and also due to the risks of pneumothorax during ventriculopleural shunt placement. If venous air embolism develops, give immediate head-down tilt and ask the surgeon to flood the surgical field with saline. If a central venous catheter is already in place air can be aspirated through the line. Children with patent foramen ovale are at risk of paradoxical air embolism.

Q. What are the current recommendations for maintaining the intraoperative PaCO₂?

Ans. Mild hypocapnia (PaCO₂ 32–35 mm Hg) following tracheal intubation may prevent further elevation of PaCO₂; however, aggressively decreasing the PaCO₂ increases the risk of brain ischemia and is not currently recommended. Normocapnia (PaCO₂ 35–45 mm Hg) should be maintained by inpatients with normal ICP.

Q. How will you monitor the child during the intraoperative period?

Ans. This procedure does not usually require invasive monitoring. Intraoperative monitoring should include:

- Electrocardiogram (ECG)
- Pulse oximeter
- Noninvasive blood pressure (NIBP) amplifier monitor
- Capnometer
- Precordial stethoscope
- Temperature monitor
- Arterial catheterization—reserved for patients with uncontrolled ICP and hemodynamic instability.

Q. How will you manage postoperative pain in this child?

Ans. Postoperative analgesia can be provided by a combination of infiltration of local anesthetic such as bupivacaine 0.25% (0.5–0.75 mL/kg) and with a combination of paracetamol (15 mg/kg) either IV or per rectum and nonsteroidal anti-inflammatory drugs (NSAIDs) if not contraindicated with additional oral opioid for breakthrough pain. High doses of long-acting opioids should be avoided because of potential detrimental effects on conscious level. Patients with severe neurological deficits can be more prone to postoperative respiratory problems;

hence, analgesics should be used judiciously and under close supervision in these patients.

Q. What are the signs and symptoms of shunt malfunction? What is the incidence of revision shunt/shunt removal and what problems can you anticipate during these procedures?

Ans. In a child with a ventriculoperitoneal shunt, the shunt is statistically unlikely to be the cause of any specific problem. However, if family members suspect shunt malfunction or no other cause for fever, malaise, behavioral change, etc., can be found (i.e., ear infection), careful and diligent evaluation of the shunt is mandatory.

Median survival of a shunt (before need for revision) in a child < 2 years of age is 2 years; > 2 years of age is 8–10 years.

The common problems of shunt malfunction may be due to obstruction or infection. Disconnections and breakage of tubing are another causes of malfunction, though less common than occlusion. Migration into the scrotum, perforation of the bowel wall, and intussusception are all rare complications in the peritoneum.

Common signs and symptoms of shunt malformation due to obstruction and infection:

Obstruction: Headache, malaise, generally not feeling well, vomiting, mental status alterations, increased BP, head circumference increase, Cushing's triad, bulging fontanelle, 6th nerve palsy signs, Macewen's sign, changes in gait, and personality changes. There may also be an increase of seizures and a complaint of neck pain. The parents often recognize signs of shunt block. Teachers may state that there has been a change of school performance.

Infection:

- Fever, meningeal signs, vomiting, signs and symptoms of shunt malfunction, abdominal pain, and peritonitis
- There may be evidence of purulent material around the shunt insertion site and redness along the shunt tract.
- Most common organisms are *Staphylococcus epidermidis* and *Staphylococcus aureus*, and also gram-negative organisms.
- External ventricular drainage may be tried in such cases. If shunt revision/removal is planned, the following points need to be kept in mind:
 - Psychological trauma of repeat surgery for parents.
 - Intravenous cannulation may be difficult due to repeat procedures.
 - Antibiotics will need to be started if infection is suspected.
 - Medical measures to decrease ICP until shunt removal.
 - In cases of shunt revision or shunt removal surgery, retrieval of the ventricular catheter may cause rupture

of the choroid plexus which has grown into the shunt lumen. This can lead to life-threatening hemorrhage.

■ SUGGESTED READING

1. Baird C, O'Connor D, Pittman T. Late shunt infections. *Pediatr Neurosurg.* 1999;31:269-73.
2. Firsching R, Schütze M, Motschmann M, Behrens-Baumann W. Venous ophthalmodynamometry: a noninvasive method for assessment of intracranial pressure. *J Neurosurg.* 2000;93:33-6.
3. Hamid RK, Newfield P. Pediatric neuroanesthesia. Hydrocephalus. *Anesthesiol Clin North Am.* 2001;19:207-18.
4. Martin LD, Bratton SL, O'Rourke PP. Clinical uses and controversies of neuromuscular blocking agents in infants and children. *Crit Care Med.* 1999;27:1358-68.
5. Nadvi SS, Du Trevoir MD, Van Dellen JR, Gouws E. The use of transcranial Doppler ultrasonography as a method of assessing intracranial pressure in hydrocephalic children. *Br J Neurosurg.* 1994;8:573-7.
6. Shemie S, Jay V, Rutka J, Armstrong D. Acute obstructive hydrocephalus and sudden death in children. *Ann Emerg Med.* 1997;29:524-8.
7. Tuli S, Drake J, Lawless J, Wigg M, Lamberti-Pasculli M. Risk factors for repeated cerebrospinal shunt failures in pediatric patients with hydrocephalus. *J Neurosurg.* 2000;92:31-8.

Meningomyelocele

Sumitra Ganesh Bakshi, Sheila Nainan Myatra

A 15-year-old child planned for surgical correction of meningomyelocele. Discuss perioperative management.

Q. What is meningomyelocele?

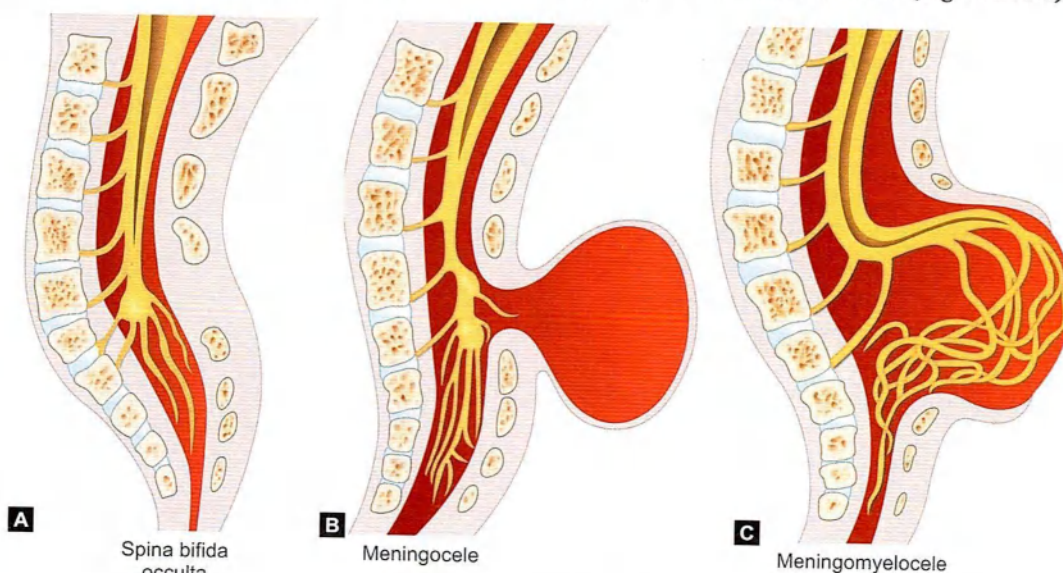
Ans. Meningomyelocele (MMC) is a congenital defect in vertebral arches with cystic dilatation of meninges and structural or functional abnormality of spinal cord or cauda equina. Meningocele on the other hand is a congenital defect in vertebral arches with cystic distension of meninges, but no abnormality of neural tissue but neurologic deficit may be present in a third of patients. Meningomyelocele is a progressive neurologic disease that eventually produces orthopedic, neurologic and genitourinary complications. Kyphoscoliosis is common in patients with thoracic lesions and it occurs in 20% of patients with lumbosacral defect.

Q. What is the pathophysiology of this condition? Discuss the embryologic basis of these defects.

Ans. The nervous system develops in two stages:

Primary neurulation: During early stage of development, the neuroepithelium bends at the midline to form neural folds which elevates, meet and fuse in midline to form the neural tube. Neural tube closure is discontinuous with distinct sites of initiation, as a result neural tube defects (NTDs) usually affects only a portion of the neural tube. The cutaneous ectoderm fuses first, to form the overlying skin, followed by the neuroectoderm. The lateral mesoderm migrates between the two ectodermal layers to form the posterior vertebral arches.

Secondary neurulation: This comprises further neural development occurring caudal to the caudal neuropore after the termination of primary neurulation. This process includes formation of the filum terminale and conus medullaris from a poorly differentiated cell mass (**Figs. 1A to C**).



Figs. 1A to C: Neural tube defects.

Meningomyelocele constitute primary NTD, which is due to primary failure of closure of neural tube or possibly disruption of already closed neural tube between 18 and 28 days of gestation. The other primary neural defects include:

- *Encephalocele*, which is an out-pouching of dura with or without brain, noted in occipital region commonly.
- *Anencephaly* is a severe form of defect not compatible with life. Cranial vault and posterior occipital bone are defective, and derivatives of the neural tubes are exposed, including both brain and body tissues.
- *Meningocele* is the herniated protrusion of only the meninges and constitutes secondary NTDs. It results from abnormal development of lower sacral or coccygeal segments during secondary neurulation. These lesions rarely are associated with hydrocephalus. The skin is typically intact over the defect. Other secondary NTDs include: sacral agenesis, dysgenesis, diastematomyelia (split cord), and myelocystocele.
- *Spina bifida occulta* is defined as failed fusion of the neural arch without herniation of the meninges or neural elements. Superficial sign of this lesion may include tuft of hair, cutaneous angiomas, lipoma or a skin dimple.

Q. What are the causes and incidence of this condition? How is the intrauterine and postnatal diagnosis made?

Ans. Meningomyelocele is more common in low socioeconomic class. Incidence is highest in Hispanics and lowest in Asians and Pacific-Islanders. Female: Male: 1.2:1. Risk increases to 2–3%, if there is one previous birth with MMC and 6–8% after two affected children.

Etiology in MMC is multifactorial.

- Specific etiologic factors are:
 - Nutritional deficiency of folic acid in mothers
 - Hypervitaminosis A
 - Associated chromosomal abnormalities—trisomies 13 and 18, single-gene mutations
 - Maternal insulin-dependent diabetes mellitus (IDDM), maternal obesity
 - Maternal hypothermia
 - Intrauterine drug exposure to valproate, carbamazepine, and ovulation inducing drugs.

Intrauterine Diagnosis

Laboratory Studies

- Elevated maternal serum α -fetoprotein levels in second trimester
- Amniotic fluid α -fetoprotein assay
- Presence of acetyl cholinesterase (a nerve-specific enzyme) in amniotic fluid.

Imaging Studies

Fetal ultrasound at about 18 weeks gestational age.

Postnatal diagnosis: Meningomyelocele is immediately obvious at birth.

After delivery MRI of spine helps in accurate assessment. Serial cranial ultrasounds, CT scan, and MRI of brain is often needed in cases of concomitant hydrocephalus.

Q. What syndromes and anomalies are associated with this condition?

Ans. Neural tube defects are often associated with anomalies of central structures which include the heart, esophagus, kidneys, brain, limbs, and anal canal. Associated anomalies which may be present:

- Club feet (neurogenic)—unilateral/bilateral (most common).
- Arnold-Chiari malformation due to associated abnormality of the cephalic anterior neural tube (80–90%).

This is the abnormality of neural tube characterized by caudal displacement of posterior fossa structures, kinking of cervicomedullary junction, the disruption of the brainstem vascular supply and lower cervical nerves. It is often associated with MMC, hydrocephalus, and syringomyelia:

- Hydrocephalus (80%)
- Neurogenic bladder (90%)
- Musculoskeletal defects (absent ribs, scoliosis)
- Urogenital anomalies—exstrophy bladder, undescended testis, hydronephrosis, solitary kidney or malformed ureters
- Hip dislocation (neurogenic)
- Facial clefts
- Anorectal malformations
- Congenital heart disease—atrial septal defect (ASD), ventricular septal defect (VSD)
- Umbilical hernia
- *VATER-L*: Vertebral, anal, tracheoesophageal fistula—fistula, renal (genitourinary), and limb anomalies. These should be diagnosed prior to the repair, because they may preclude a long-term outcome.

Q. What are the common clinical findings in this condition?

Ans.

- Paraplegia
- Hydrocephalus
- Cranial nerve dysfunctions
- Seizures
- Neurogenic bladder and bowel
- Renal failure
- Progressive bony, spine and joint deformities
- Pathological fractures.

Q. What specific history will you ask for in a patient with MMC? How will you proceed with the examination in these patients prior to corrective surgery?

Ans. Specific history includes:

- Antenatal history of drug ingestion
- Similar defect in siblings

- Birth history—prolonged labor and fetal hypoxia can occur in babies with large head due to hydrocephalus
- History to rule out associated symptoms and signs like seizures
- Increased precordial activity, found in associated congenital heart disease
- Limb movements to rule out neurological deficits.

General assessment of the newborn:

- For overall development and nutrition
- Look for deformities, evidence of muscle weakness, and contractures.

Back examination:

- Inspect the defect—its location, size and shape, and any cerebrospinal fluid leak.
- Note the curvature of spine.
- Palpate for deformity like spina bifida which is generally associated with MMC. One should exercise due caution while palpating.
- Latex allergy is known and it is preferable to use a sterile non-latex gloves while examining the sac.

Neurological examination:

- Observe for spontaneous activity to sensory stimuli in all extremities
- Tendon reflexes could often be absent in associated neurological deficits
- Signs of associated hydrocephalus:
 - Increase in the head circumference
 - Bulging of the anterior fontanel
 - Cranial nerve involvement abducent nerve paresis leading to conjugate movement abnormalities.

Airway assessment:

- Rule out associated facial cleft
- In children with Arnold-Chiari syndrome—inspiratory stridor, and apneic episodes is common.

Associated congenital anomalies (to be ruled out):

- Kidney enlargement
- Club feet
- Hip dislocation
- Cardiomegaly.

Q. What investigations will you ask for in this patient?

Ans. Investigations needed include the routine baseline and specific (neurological) investigations. These include:

- Hemoglobin and platelet count
- White cell count could be elevated in cases of infection which could be cause of an infected sac, at times UTI are also seen
- Serum creatinine
- Urine routine and microscopy—important in this case as various associated anomalies make the child susceptible to repeated episodes of urinary tract infection.
- X-ray chest may be asked if child is symptomatic. Also useful to rule out cardiomegaly.

- *Blood grouping and cross-matching:* With large defect, blood loss may be significant and may warrant transfusion.
- CT brain and MRI—to evaluate the presence of hydrocephalus. With hydrocephalus MRI shows characteristic appearance, i.e. occipital horns are more dilated than the frontal horns, and the long-axis of left ventricle tends to be parallel.
- As MMC may be associated with involvement of other organ systems, additional investigations will be warranted as per the presence of this involvement:
 - *2D echocardiography:* To rule out congenital heart disease.
 - *Ultrasound of urinary tract:* To detect possible hydronephrosis and structural abnormalities of urinary tract.
 - Urodynamic studies—such as voiding cystourethrogram.

Q. Describe the management of child with meningo-myelocoele? What are the surgical options for this condition?

Ans. Management of a child with MMC includes the following three aspects:

1. *Assessment and management of lesion:*
 - If lesion has ruptured start antibiotics
 - Cover the lesion with sponges soaked in NS or RL to prevent desiccation
 - Trendelenburg position, with child on stomach (keeps pressure of lesion)
 - Monitor infant closely for signs of meningitis.
2. *Neurological assessment:*
 - Assess and document presence of neurodeficit.
3. *Ancillary assessment and management:*
 - Evaluation by neonatologist to assess other abnormalities
 - Urological consultation
 - Orthopedic consultation for severe kyphotic or scoliotic spine deformities and hip or knee deformities.

Q. What are the surgical considerations in MMC patients?

Ans. Surgery in utero is now available at many centers. The benefit of early correction is being evaluated at many centers.

In case an early diagnosis is made and in utero correction has not been done, then the pediatric team should be present for delivery, which should almost always be by cesarean section since these patients are at a high risk of developing latex allergies, which can be quite severe and life threatening. Therefore, attempts should be made to avoid latex exposure and sensitization.

Timing of closure: Early closure of MMC defect is not associated with improvement of neurological function, but

leads to lower infection rates. The defect should be closed within 24 hours, whether or not membrane is intact.

Simultaneous repair and ventriculoperitoneal (VP) shunting: In patients with clinically overt hydrocephalus at birth, MMC repair and VP shunt may be done in the same sitting without increased risk of infection. If VP shunt is not performed, an increased risk of MMC repair breakdown is present.

Outcomes: With modern treatment 85% of infants survive. Mortality is mainly related to associated conditions such as Arnold-Chiari syndrome, shunt malfunction, and infections. About 80% patients will have normal IQ.

Q. What are the problems anticipated in the perioperative period?

Ans.

- *Problems related to age:*
 - Mainly a neonate/infant
- *Problems related to airway:*
 - Large head (if associated hydrocephalus)
 - Associated facial clefts
 - Pediatric airway and its implications
- *Problems related to other associated syndromes/anomalies:*
 - Hydrocephalus
 - Arnold-Chiari syndrome, often presents with stridor
 - Congenital heart disease
 - Renal problems
 - Musculoskeletal disorders, succinylcholine should be avoided in cases of muscle weakness
- *Problems related to surgery:*
 - Surgery in prone position:
 - Extreme head flexion in Chiari malformation may cause brainstem compression
 - Improper positioning may lead to venous congestion of face, tongue, and neck and reduce lung compliance
 - Increase abdominal pressure causes vena caval compression that can cause increase bleeding through engorged epidural veins
 - Often surgery dissection could be difficult in case of tethered cord
 - Hypothermia
 - Blood loss can be difficult to assess in view of simultaneous cerebrospinal fluid (CSF) loss
 - Nerve studies may be needed intraoperatively to help in identification in few cases
 - At dural closure Valsalva maneuver is needed to test the integrity of closure
- *Problems in the postoperative period:*
 - Due to nursing in prone position

- *Acute hydrocephalus:*

- If not present preoperatively, can occur due to closure of defect
- May occur due to shunt malfunction.

Q. How will you premedicate this child? How will you induce anesthesia?

Ans. The need to premedicate will depend upon the age of the child. Since defect correction is done early, the neonate will not need anxiolytics for premedication. Antisialagogue may be given as the patient will be placed prone. Blood sugar monitoring would be needed. In case if the parents bring the child later in infancy, which is not uncommon in our country—premedication, to allay separation anxiety, will be needed. The choice will depend upon presence of associated raised ICT. In such cases, ketamine (oral/intramuscular) is best avoided and oral midazolam is preferred.

In 1995, Viscomi et al. reported a series of 14 neonates will lumbar or sacral MMC which were done under spinal anesthesia. However, general anesthesia remains the technique of choice as the surgery is in prone and airway remains secure.

Induction of General Anesthesia

- In syndromic babies and abnormal facies, a difficult airway is anticipated. The induction agent of choice would be inhalational anesthetic. Sevoflurane is preferred. Intubation with difficult airway kit should be ready and spontaneous ventilation is safest.
- In patients with neurological deficit succinylcholine should be avoided.
- In babies with a huge defect placement of the child during intubation is important.
- The MMC should be placed with adequate padding and the rest of upper part of the body could rest on an adequate size pillow.
- Alternatively the patient can be administered anesthetics and trachea intubated in the lateral position.

Q. How is the child positioned? How will you maintain anesthesia and monitor this child during surgery?

Ans. The child is positioned prone on adequate sized bolster and head well-supported all bony points and eyes are padded and abdomen should be free. Anesthesia is maintained with inhalational agents and opioids for pain relief. Use of relaxant should be timed properly in case if nerve studies are needed for nerve identification intraoperatively.

Monitoring: Routine monitoring that is used includes ECG, pulse oximetry, capnometry, noninvasive blood pressure, and invasive BP which may be needed in patients with large defects and difficult anatomy. Temperature monitoring, hourly urine output, and careful and accurate measurement of blood loss are required.

Q. What are the anesthetic implications for subsequent surgery in this child?

Ans. Due to multisystem involvement the patient may need repeated surgeries for the following:

Hydrocephalus: Child may need VP shunt; if the shunt is already done, it may require revision procedure.

If the patient is undergoing tethered cord correction, muscle relaxation may have to be withheld at the time of motor function assessment. The child may need orthopedic intervention later for correction of skeletal deformities. Scoliosis deteriorates in the presence of untreated hydrocephalus and improves following successful shunting in patients with MMC. Neurogenic bladder which does not respond to clean intermittent catheterization (CIC) and/or medications may require surgical intervention for augmentation cystoplasty and urinary diversion to prevent renal failure. The number of surgical interventions and atopic disposition lead to development of latex allergy in these children. Establishment of latex-free environment for surgery is a must. Children with MMC often also have nerve problems and dysfunction of the bowels, known as neurogenic bowel. They will often have poor or weak bowel peristalsis and motility. Malnutrition is commonly seen in such children.

Q. What is the role of fetal surgery in these patients? What are the considerations for the mother and fetus during fetal surgery?

Ans. Recently patients with severe MMC and other anomalies have been treated in utero. The neurological outcomes are better than surgeries done during neonatal period. Essentially fetal surgery can be of the following types:

- Minimally invasive (e.g., placental blood sampling, insertion of shunt).
- Percutaneous fetoscopic surgery (e.g., fetoscopic endoluminal tracheal occlusion (FETO) in fetus with impaired lung development as seen in congenital diaphragmatic hernia).
- Fetoscopic surgery through maternal laparotomy (recommended for MMC repair).
- Open surgery involving hysterotomy.
- Ex utero intrapartum treatment (EXIT) procedure usually done near term. This generally involves securing the fetal airway while the fetus is still being oxygenated by the placental circulation. The fetal airway is secured and oxygen is maintained (by extracorporeal membrane oxygenator, if needed) before the placenta is clamped. This is important in fetus who are unable to oxygenate on delivery due to airway abnormalities.

Maternal Considerations

The choice of anesthetic technique depends on the nature of the surgery. For minimal invasive procedures, regional techniques are preferred while for open fetal surgery or EXIT

procedure general anesthesia to the mother is the technique of choice. The maternal concerns are similar to that for a pregnant patient undergoing non-obstetric surgery (See Chapter 15 "Non-obstetric Surgery in a Pregnant Patient"). *Additional factors include:*

- Teratogenic effects of anesthetic drugs (refer Chapter 15, Nonobstetric Surgery in a Pregnant Patient).
- Effect of anesthetic drugs on developing brain. The negative effects of anesthetic agents on developing brain has been a matter of concern. Literature suggests anesthetic drugs increase the neurodegeneration, and reduce neurogenesis in animals. Direct evidence in humans is lacking and is currently a topic for future research.
- *Ensuring adequate fetal anesthesia:* It has been shown that fetus also experiences pain and develops stress in response to the operative procedure. Intravenous remifentanyl infusion given to mother crosses placenta and can be used to titrate to fetal pain relief. Fetal analgesia and muscle relaxation can also be obtained by direct placental or intramuscular injections of opioids and muscle relaxants.
- *Monitoring for postoperative complications:* One must monitor for potential complications including hemorrhage, premature rupture of membrane, chorioamniotic membrane separation, premature labor, and fetal distress.

SUGGESTED READING

1. Brustia L, Preseglio I, Adduci D, Imberti R, Fanzio M, Cimino F, et al. Anesthesia in the surgical repair of myelomeningocele. *Minerva Anesthesiol.* 1993;59:853-6.
2. Gupta R, Kilby M, Cooper G. Fetal surgery and anaesthetic implications. *Contin Educ Anaesth Crit Care Pain.* 2008;8:71-5.
3. Husain AM, Shah D. Prognostic value of neurophysiologic intraoperative monitoring in tethered cord syndrome surgery. *J Clin Neurophysiol.* 2009;26:244-7.
4. Kabagambe SK, Jensen GW, Chen YJ, Vanover MA, Farmer DL. Fetal Surgery for Myelomeningocele: A Systematic Review and Meta-Analysis of Outcomes in Fetoscopic versus Open Repair. *Fetal Diagn Ther.* 2018;43:161-74.
5. Soundararajan N, Cunliffe M. Anaesthesia for spinal surgery in children. *Br J Anaesth.* 2007;99:86-94.
6. Tulipan N. Intrauterine closure of myelomeningocele: an update. *Neurosurg Focus.* 2004;16:E2.
7. Van de Velde M, De Buck F. Fetal and maternal analgesia/anesthesia for fetal procedures. *Fetal Diagn Ther.* 2012;31:201-9.
8. Vercauteren M, Palit S, Soetens F, Jacquemyn Y, Alahuhta S. Anaesthesiological considerations on tocolytic and uterotonic therapy in obstetrics. *Acta Anaesthesiol Scand.* 2009;53:701-9.
9. Viscomi CM, Abajian JC, Wald SL, Rathmell JP, Wilson JT. Spinal anesthesia for repair of meningomyelocele in neonates. *Anesth Analg.* 1995;81:492-5.
10. Wu D, Ball RH. The maternal side of maternal-fetal surgery. *Clin Perinatol.* 2009;36:247-53.

Traumatic Brain Injury

Atul Prabhakar Kulkarni, Malini Joshi, Sudivya Sharma

A 35-year-old male patient presents to the casualty with history of vehicular accident. He was conscious at the site of accident and became progressively drowsy during transport. At presentation in the casualty, he has become unresponsive and has no purposeful activity. He has multiple bruises over the right side of chest and abdomen and he appears to be in respiratory distress.

His pulse rate is 130 beats/min, blood pressure is 96/68 mm Hg, and saturation is 93% on room air.

Q. How will you assess this patient?

Ans. Initial management consists of rapid initial assessment and simultaneous institution of treatment along with necessary imaging.

Advanced trauma life support (ATLS) scheme to assess trauma patient:

- Preparation and triage
- Primary survey—ABCDE
- Immediate resuscitation of patients with life-threatening injuries
- Adjuncts to primary survey and resuscitation
- Consider need for patient transfer
- Secondary survey (head to toe examination and history of patient)
- Adjuncts to secondary survey
- Continued postresuscitation reevaluation and monitoring of the patient
- Definitive care.

The *prehospital phase* emphasizes on airway control, immediate control of external bleeding and shock, immobilization of patient, immediate notification, and transfer to hospital with minimal scene time.

The *hospital phase* involves advance planning for patients arrival, with appropriate airway equipment and monitoring devices, warm intravenous crystalloid fluids, and appropriate medical assistance. It involves:

A—Perform visual scan of patient for obvious injuries

B—Obtain history from prehospital personnel and patient

C—Primary survey (ascertain “ABCDEs”) with simultaneous resuscitation.

Airway maintenance with (cervical spine control): Look for foreign bodies and fractures (facial, mandibular, tracheal, etc.), assess Glasgow coma scale (GCS) score with placement of definitive airway if score < 8. Ensure avoidance of cervical spine movements. Establish surgical airway if intubation is contraindicated or cannot be established ATLS guidelines, 10th edition, page 61.

Breathing (give supplemental oxygen): Look for chest wall movements, retraction, and nasal flaring. Determine whether ventilation is adequate, and listen for stridor, and obstructed ventilation. Provide assisted ventilation for ventilator failure and inspect chest to exclude open pneumothorax, sucking chest wound or flail segment.

Circulation (establish venous access): Check peripheral pulses, grade shock according to vital signs, correct hypovolemia, and obtain blood samples.

Disability (determine neurologic status): Evaluate central function (AVPU):

A—Alert, V—Responds to vocal stimulus, P—Responds to painful stimulus, and U—Unresponsive.

Evaluate pupillary response to light.

Expose patient for complete examination:

D—Resuscitation phase

E—Secondary survey

F—Definitive care phase.

Q. How is the central nervous system (CNS) assessed? Does this patient need to be intubated? How will you intubate him?

Ans. Central nervous system is assessed using the Glasgow Coma Score (GCS scale) or the AVPU scale. GCS scale,

ranges from 3–15, if <8; then the patient has serious traumatic brain injury with raised intracranial pressure (ICP > 20 mm Hg). The GCS score is given in **Table 1**.

TABLE 1: Revised Glasgow coma scale.

<i>Eye opening (1–4):</i>	
Spontaneous	4
To speech (not necessarily a request for eye opening)	3
To pressure	2
None	1
Nontestable	NT
<i>Best motor response (1–6):</i>	
Obeys commands	6
Localizes purposeful movement toward the stimulus	5
Normal flexion (withdraws from painful stimulus)	4
Abnormal flexion (decorticate posture)	3
Extension (decerebrate posture)	2
No movement	1
Nontestable	NT
<i>Verbal response (1–5):</i>	
Oriented (knows name, age)	5
Confused (still answers questions)	4
Words	3
Sounds	2
None	1
Nontestable	NT
Severe head injury <8, moderate head injury 9–12, mild head injury 13–15, NT—element which is nontestable.	

Factors such as hypoxia, hypotension, and alcohol intoxication affect GCS. Therefore, the patient should be resuscitated and reversible causes corrected before GCS assessment.

GCS-pupils (GCS-P) is scored from 1 to 15, calculated by subtracting pupil reactivity score (PRS) from the calculated GCS. Pupil reactivity score - both pupils unreactive to light = 2, one pupil unreactive to light = 1, both pupils are reacting = 0.

GCS-PA (GCS, pupils, age and CT findings), a predictive score useful for prognostication but is not easily applicable at the bedside and also difficult to interpret therefore has not been accepted worldwide.

Patients who are not alert and not responding to command (P or worse) are equivalent to a GCS of around 8 which indicates a severe injury.

This patient appears to be maintaining his airway and is maintaining his saturation. However, the ATLS and Brain Trauma Foundation (BTF) guidelines recommend that at GCS < 8; patients should be intubated for airway protection, therefore, this patient should be intubated. Rapidly worsening GCS, presence of hemorrhagic shock,

and need to transport patients for imaging or surgery should also prompt intubation and ventilation. Cervical spine protection and restriction of motion is of primary importance during intubation. Patient is assumed to have cervical spine fracture at all times and it is protected with hard cervical collar until the spine is cleared radiologically. Orotracheal intubation is performed while ensuring manual inline stabilization and cervical spine motion restriction. It is performed by an assistant crouching at the head of table grasping the mastoid processes while the front part of the collar is removed to allow adequate mouth opening.

The usual induction drugs include an intravenous narcotic (fentanyl 100–150 µg, pethidine 50 mg or morphine 5 mg) followed by a reduced dose of thiopentone (150–200 mg) or propofol (90–100 mg) to ensure anesthesia, without causing hypotension. Intubation technique will need to be modified in case of maxillofacial trauma or in presence of fracture of the mandible. The use of videolaryngoscopes in intubation is also recommended by ATLS guidelines. Surgical airway; preferably surgical cricothyroidotomy (in desperate situations) or surgical tracheostomy (without giving neck extension) may be considered in anticipated difficult intubation in the absence of videolaryngoscopes, to avoid chances of exacerbating possible cervical spine injury. The Ryle's tube should be placed via the orogastric route in such patients.

All trauma patients are assumed to be full stomach during intubation. A drug assisted intubation which includes modified rapid sequence intubation is done in patients with intact gag reflexes, especially in patients who have sustained head injuries. Avoid nasal endotracheal intubation to prevent cerebral infection as basilar skull fracture associated with cerebrospinal fluid (CSF) leak, cranial nerve injuries (vestibulocochlear, olfactory), and intracranial air may be present.

Q. How will you assess and replace the blood loss in this patient?

Ans. Hemorrhage is the predominant cause of preventable deaths after injury. External hemorrhage is managed by direct manual pressure, tourniquets, and hemostatic agents. Internal hemorrhage could arise from chest, abdomen, retroperitoneum, pelvis, and long bones. Chest X-ray, pelvic X-ray, and abdominal assessment with either extended focused assessment sonography in trauma (e-FAST) or diagnostic peritoneal lavage (DPL), and bladder catheterization may be done to assess hemorrhage in these patients. Management includes chest decompression, pelvic binders, splint application, and surgical intervention.

The blood loss can be assessed from the vital signs of the patients. Initially a bolus of one liter isotonic fluid for adults and 20 mL/kg for pediatric patients <40 kg may be given for resuscitation. Continuous resuscitation with

>1 L crystalloids is avoided due to reports of increased mortality. For ongoing blood losses >30%, blood and blood products should be transfused without delay. Balancing the goals of organ perfusion with the risks of rebleeding by accepting lower than normal blood pressure has been termed as “balanced resuscitation.” It involves transfusion of minimal crystalloids while giving plasma, platelets, and red blood cell transfusion early at a ratio of 1:1:1 and allowing permissive hypotension with an aim to prevent coagulopathy and thrombocytopenia. If brain injury is suspected, avoid hypotension to minimize secondary brain injury and optimize intracranial hemodynamics. Use rotational thromboelastometry (ROTEM) (ATLS guidelines, 10th edition, page 109) or thromboelastography (TEG) in resuscitation to reverse and prevent coagulopathy. In pediatric population, weight-based blood product resuscitation with 10–20 mL/kg of RBC and 10–20 mL/kg of fresh frozen plasma and platelets should be given. Uncrossed O Rh-negative blood can be used when urgent and then appropriately crossmatched blood is given once the patient’s blood group is obtained. Tranexamic acid 1 g over 10 minutes stat given within 3 hours of trauma and 1 g over 8 hours can also be considered.

If more than four units of blood required, prepare for massive transfusion protocol. Assessment of blood composition score (ABC score) for trauma patients predicts need for massive transfusion. It includes scores for injury by penetrating mechanism, positive FAST, BP $\leq$ 90 mm Hg, and heart rate > 120 beats/min. Each positive finding is given a point of 1. Scores <2 are unlikely to require massive transfusion. If uncontrolled bleeding present, take detailed history for antiplatelet or anticoagulant medications, administer reversal agents, and TEG or ROTEM monitoring for blood products.

A hemodynamically stable patient may have persistent tachycardia, tachypnea, and oliguria; this patient is clearly under-resuscitated and still in shock (Table 2).

TABLE 2: Signs and symptoms of hemorrhage by class.

	Class I	Class II (mild)	Class III (moderate)	Class IV (severe)
Vital signs	<15%	15–30%	30–40%	>40%
Heart rate	$\leftrightarrow$	$\leftrightarrow/\uparrow$	$\uparrow$	$\uparrow/\uparrow\uparrow$
SBP	$\leftrightarrow$	$\leftrightarrow$	$\leftrightarrow/\downarrow$	$\downarrow$
Pulse pressure	$\leftrightarrow$	$\downarrow$	$\downarrow$	$\downarrow$
Urine output	$\leftrightarrow$	$\leftrightarrow$	$\downarrow$	$\downarrow\downarrow$
Capillary refill	Normal	Delayed	Delayed to absent	Absent
Base deficit	0 to –2 mEq/L or less	–2 to –6 mEq/L	–6 to –10 mEq/L	–10 mEq/L

Contd...

	Class I	Class II (mild)	Class III (moderate)	Class IV (severe)
Respiratory rate	$\leftrightarrow$	$\leftrightarrow$	$\leftrightarrow/\uparrow$	$\uparrow$
Mental status GCS	Anxious $\leftrightarrow$	More anxious $\leftrightarrow$	Anxious and confused $\downarrow$	Confused and lethargic $\downarrow$
Whether blood products are required	Monitor	May be possible	Yes	Massive transfusion protocol

(GCS: Glasgow coma scale; SBP: systolic blood pressure).

Q. What are the adjuncts to primary survey?

Ans. Electrocardiographic monitoring, urinary and gastric catheters (avoid nasogastric tubes if base skull fracture is suspected), ventilatory rate and arterial blood gases, pulse oximetry, blood pressure and X-ray examinations and diagnostic studies (e-FAST and DPL).

Q. What do you mean by e-FAST?

Ans. e-FAST is used in unstable trauma and includes the following views:

- Pericardial view
- Right upper quadrant (RUQ) view to include diaphragm—liver interface and Morrison’s pouch
- Left upper quadrant (LUQ) view to include diaphragm—spleen interface and spleen-kidney interface
- Suprapubic view
- LUQ and RUQ view with attention on diaphragm and adjacent lung to evaluate hemothorax
- Evaluation of pneumothorax with probe placed in 2nd intercostal space in midclavicular line.

Q. What is diagnostic peritoneal lavage?

Ans. It indicated in unstable blunt trauma or penetrating trauma. Infra-umbilical insertion of peritoneal dialysis catheter either by incision or Seldinger technique by using 18-G needle is done. A *positive test*—aspiration of gross blood >10 mL, *indicates* need for surgical intervention. If grossly negative, then infusion of 1 L normal saline through catheter is done, DPL is considered positive if >100,000 RBCs/mm³ or >500 WBCs/mm³ or positive Gram stain for food fibers or bacteria. A negative lavage does not exclude retroperitoneal injuries, such as pancreatic and duodenal injuries.

Q. What is primary brain injury? What are secondary injuries? Why are these important?

Ans. Primary brain injuries result immediately from the initial trauma. It includes contusion, damage to blood vessels and axonal shearing in which the axons are stretched

Contd...

and torn. The blood-brain barrier and meninges may be damaged causing neuronal death. Secondary injury results in further brain damage from ischemia, low blood pressure, and cerebral swelling. This results from factors such as hypoxia, high carbon dioxide levels, and venous congestion. Prolonged hypotension and hypoxia increases mortality in head injury patient by 70–80%. The primary goal of treatment is to prevent secondary brain injury. A few examples of primary and secondary injuries are given in **Table 3**.

TABLE 3: Primary and secondary brain injury.

Primary brain injury	Secondary brain injury
• Intracerebral hemorrhage • Subdural hemorrhage • Subarachnoid hemorrhage • Epidural hemorrhage • Cerebral contusion • Cerebral laceration • Axonal stretch injury	• Edema • Impaired metabolism • Altered cerebral blood flow • Free radical formation • Excitotoxicity • Hypoxia, hypotension, hyper- or hypocapnia, hyper- or hypoglycemia, hydrocephalus, intracranial hypertension, vasospasm, metabolic derangement, calcium, ions toxicity

Q. What is Canadian C-spine rule and National Emergency X-radiography Utilization Study (NEXUS) criteria?

Ans. It is a clinical decision tool for cervical spine evaluation. In Canadian C-spine rule, radiography is mandated if:

- Age > 65 years.
- Dangerous mechanism of injury
- Paresthesia in extremities.

In National Emergency X-radiography Utilization Study (NEXUS) criteria, radiography is not required if:

- No posterior midline cervical-spine tenderness
- No evidence of intoxication
- A normal level of alertness
- No focal neurologic deficit
- No painful distracting injuries.

These patients are extremely unlikely to have an acute C-spine fracture or instability.

Q. What are the indications of CT scan in head injury patients?

Ans. Witnessed loss of consciousness, definite amnesia, or witnessed disorientation in a patient with GCS of 13–15, GCS <15 at 2 hours after injury, basilar skull fracture (e.g., hemotympanum, CSF otorrhea or rhinorrhea, Battle's sign), vomiting (more than two episodes), age >65 years, anticoagulant use, dangerous mechanism (e.g., pedestrian struck by motor vehicle, occupant ejected from motor vehicle, fall from height >3 feet or five stairs), moderate brain injury (GCS <9). A shift of 5 mm or greater often indicates the need for surgery for evacuation of blood clot or contusion.

Q. What is excitotoxicity?

Ans. Head injury leads to release of glutamate from neuronal cells causing increased concentration of glutamate in CSF leading to increase in intracellular calcium ion. This further causes activation of protein kinase, phospholipase, nitric oxide synthase, proteases, and other enzymes causing proteolysis, free radical formation, lipid peroxidation, deoxyribonucleic acid (DNA) damage, and ultimately neuronal death.

Q. How is the head injury patient resuscitated?

Ans. It is very important to rapidly achieve cardiopulmonary stabilization in patients with severe brain injury:

- **Airway:** Early endotracheal intubation as transient respiratory arrest and hypoxia causes secondary brain injury.
- **Ventilation:** Aim at $\text{SaO}_2 > 98\%$ and PaCO_2 around 32 mm Hg.
- **Cardiovascular stability:** Maintain SBP ≥ 100 mm Hg for patients 50–69 years or at ≥ 110 mm Hg or higher for 15–49 years or older than 70 years.
- Lower ICP.
- Maintain cerebral perfusion pressure (CPP = MAP – ICP). Vasopressors can be added to increase mean arterial pressure (MAP).

Q. What is cerebral perfusion pressure (CPP)? What are the factors affecting CPP?

Ans. The cranium, a rigid box, contains brain (80%), CSF (8%), and blood (12%). Increase in the amount of any of these will lead to increased ICP since the cranium cannot expand. This in turn will affect the cerebral blood flow (CBF), as the CPP is dependent on the MAP and the ICP. Normal ICP is about 5–13 mm Hg (**Fig. 1**). Pressures >22 mm Hg, particularly if sustained and refractory to treatment are associated with poor outcomes.

When there is an increase in the volume of either the brain or blood, the CSF is forced out into the spinal sac maintaining the ICP. With further increase in intracranial volume, venous blood and more CSF is forced out of the skull. When these compensatory mechanisms fail, there is an abrupt increase in ICP. Increase in the ICP and/or a fall in MAP or a combination of both will lead to a situation where CPP becomes very low; leading to progressive decrease in CBF ultimately causing brain death.

Assessment of CPP is vital and possible either by measurement of both ICP and MAP or by measuring MAP and making a reasonable estimate of ICP. During anesthesia, if ICP is raised, a fall in blood pressure must be avoided or treated quickly by volume replacement or vasopressors. When CPP is inadequate, the oxygen saturation of jugular venous blood falls (normal range 65–75%) due to increased oxygen extraction. Inadequate CPP (<70 mm Hg)

contributes to poor outcomes in patients with raised MAP > 90 mm Hg.

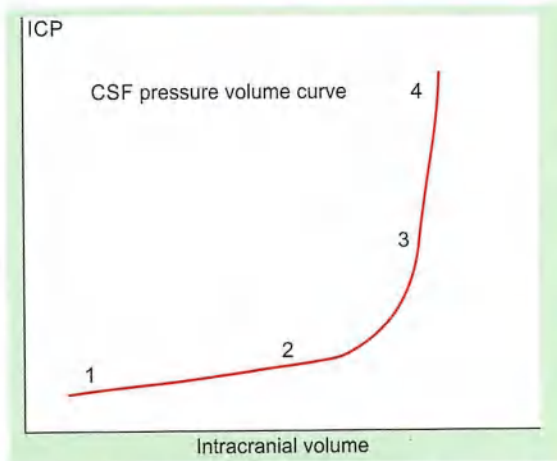


Fig. 1: Monro–Kellie doctrine: stage 1–2 are compensation phase, stage 3–4 are decompensated phase. (CSF: cerebrospinal fluid).

Q. Discuss in brief about cerebral blood flow (CBF) and factors affecting CBF.

Ans.

Cerebral blood flow: The normal CBF is 45–50 mL/100 g/min ranging from 20 mL/100 g/min in white matter to 70 mL/100 g/min in gray matter. In normal circumstances when the flow falls to <18–20 mL/100 g/min, physiological electrical function of the neuronal cell begins to fail. Also, an increase or decrease in CBF causes an increase or decrease in cerebral arterial blood volume because of arterial dilatation or constriction. Thus, in a brain which is decompensated as a result of major intracranial pathology, increases or decrease in CBF will in turn lead to a significant rise or fall in ICP.

Autoregulation: Cerebral blood flow is maintained at a constant level in normal brain in the face of the usual fluctuations in blood pressure by the process of autoregulation. It is a poorly understood local vascular mechanism. Normally autoregulation maintains a constant blood flow between MAP 50 and 150 mm Hg. However, in traumatized or ischemic brain, or following vasodilator agents (volatile agents and sodium nitroprusside); CBF may become blood pressure dependent. Thus, as arterial pressure rises, CBF will rise, causing an increase in cerebral volume. Similarly as pressure falls, the CBF will also fall, reducing ICP, but also inducing an uncontrolled reduction in CBF. If CPP falls below the critical value of 70 mm Hg, the patient will have inadequate cerebral perfusion. Autoregulation will cause cerebral vasodilatation leading to a rise in brain volume. This in turn will lead to a further rise in ICP and induce the vicious circle described by the vasodilatation cascade resulting in cerebral ischemia.

Carbon dioxide causes cerebral vasodilatation. As the arterial tension of CO_2 increases, CBF increases and when CO_2 levels fall, vasoconstriction is induced. Hyperventilation can lead to a mean reduction in ICP of about 50% within 2–30 minutes; however, when PaCO_2 is <25 mm Hg (3.3 kPa), there is no further reduction in CBF. Hence, there is no advantage in inducing further hypocapnia; as it only shifts the oxygen dissociation curve further to the left, making oxygen less available to the tissues. Acute hypocapnic vasoconstriction will only last for a relatively short time (5 hours). While hypocapnia is maintained, there is a gradual increase in CBF toward control values leading to cerebral hyperemia (overperfusion). When long-term ventilation is required, only mild hypocapnia (34–38 mm Hg; 4.5–5.1 kPa) should be maintained.

Q. What is the triphasic response to head injury?

Ans. The triphasic response to head injury is a sequential derangement of water balance seen postneurosurgery, particularly in pituitary surgery patients. Patients display polyuria (due to diabetes insipidus with rising sodium), followed by oliguria [due to syndrome of inappropriate antidiuretic hormone secretion (SIADH) and low sodium] followed lastly by a return to normalcy or a polyuric response again. This triphasic response may be due to hypothalamic dysfunction, later release of vasopressin from the damaged pituitary and, lastly due to the exhaustion of vasopressin.

Q. The patient was intubated in casualty and mechanically ventilated. CT scan of the head showed right temporoparietal extradural hematoma. Patient is now posted for evacuation of extradural hematoma. Discuss the intraoperative management of this patient. How will you manage hemodynamics of this patient? What monitoring will you need?

Ans.

Hemodynamic management: This patient is already intubated in the casualty. However, if the patient is intubated in OT, avoid depression of the cardiovascular system and hypotension by anesthetic drugs and initiation of intermittent positive-pressure ventilation (IPPV) as it leads to reduced CBF and oxygenation causing ischemia and major neurological damage. Hypotension (SBP < 90 mm Hg) being an independent predictor of poor outcomes after head trauma is prevented by preloading with saline or rapid fluids after anesthetic induction. Small doses of vasopressor (noradrenaline) infusion can be started and titrated to support blood pressure and maintain CPP of at least 70 mm Hg. If the MAP is low such that the CPP < 70 mm Hg, autoregulation leads to arterial dilatation in an attempt to reduce the resistance to increased flow. This leads to further increase in brain volume and ICP, worsening the situation.

Also, hypertension might be undesirable if autoregulation is impaired, as it leads to increased CBF and bleeding.

Monitoring: It includes electrocardiography, pulse oximetry, temperature and end-tidal CO₂ monitoring. Continuous invasive arterial pressure monitoring is useful for monitoring CPP, and titration of vasopressors and fluids. A central venous access helps in assessing volume status as well allows infusion of vasopressors. Surgery should not be delayed while taking time for institution of invasive monitoring. ICP monitoring is extremely useful and an intraventricular drain placed during surgery can be used for monitoring as well as for CSF drainage to decrease ICP. Urine output needs to be monitored as a surrogate for perfusion and also since mannitol is used to control ICP.

Maintenance of anesthesia: Anesthesia is maintained using narcotics (IV fentanyl or morphine given slowly), vecuronium and the patient ventilated with inhalational agents (isoflurane—first choice) in oxygen enriched air. Most intravenous drugs decrease metabolism and maintain coupling excluding ketamine. High inspired concentrations of inhalational agents [>1 minimum alveolar concentration (MAC)] will cause cerebral vasodilatation with increased cerebral blood volume, cerebral edema and consequent rise in ICP. They decrease metabolism and change metabolic coupling and can cause hypotension. This combination of raised ICP and low BP is dangerous as it leads to reduced CPP, worsening neuronal damage and adverse outcomes. Nitrous oxide is contraindicated in patients with raised ICP.

Ventilatory strategy: Aim is to provide adequate oxygenation, avoid hypoxemia and prevent sudden increase in CBF and ICP, with targets of low normal PaCO₂ (approximately 35 mm Hg), reduction of cerebral swelling and ICP.

Q. Describe hyperosmolar therapy in neurosurgical patients?

Ans. Mannitol and hypertonic saline are two main drugs used for decreasing ICP. Mannitol is available as 20% w/v solution in 100 mL bottles, containing 20 g/100 mL. It has two mechanisms of action, one is its immediate plasma expanding effect leading to reduced hematocrit, increased erythrocytes deformability, decreased blood viscosity, and increased CBF and oxygen delivery. This rheological effect takes few minutes to set in. It also reduces cerebral parenchymal cell water. The osmotic effect takes 15–30 minutes while gradients are established between plasma and cells. It lasts for 90 minutes to 6 hours or more.

Hypertonic saline acts by osmotic mobilization of water across intact blood–brain barrier which reduces cerebral water content. It dehydrates endothelial cells and erythrocytes which increases the diameter of vessels and

improves blood flow. It also reduces leukocyte adhesion in traumatized brain. The rebound phenomenon reported with mannitol is not present in hypertonic saline in traumatic brain injury. But, exclude hyponatremia prior to administering hypertonic saline as it bears the risk of cerebral pontine myelinolysis. Also, it can aggravate pulmonary edema in patients with underlying cardiac or pulmonary problems.

Q. How will you manage fluid therapy in a head injured patient?

Ans. The brain is surrounded by a membrane separating it from the vascular space (blood–brain barrier), which allows only water to pass through it. Avoid hypotonic fluids such as dextrose solutions in water (5% dextrose) as dextrose metabolism leaves just the water or a very dilute saline solution which is taken up by the brain causing cerebral edema and raised ICP. 0.9% normal saline has a similar concentration of sodium and is the fluid of choice. Avoid hypovolemia and fluid overload, give colloids and blood products to treat hypovolemia due to major blood loss.

Q. What are the predictors of outcome in head injury patients?

Ans.

- Age
- *CT scan diagnosis:* Marshall scale and Rotterdam scale are used to predict the risk of increased ICP and outcome in adults based on increasing severity on the basis of findings on noncontrast brain CT scan
- GCS score at admission
- Pupillary activity
- Hypotension.

Q. If he had liver laceration with significant intra-abdominal bleeding as well, what should be done first evacuation of cranial blood or laparotomy?

Ans. Both would be evacuated at the same sitting, however, evacuation of cranial blood is the foremost priority as there are higher chances of developing secondary injury due to increasing intracranial volume of blood, higher ICP, thus lower CPP and cerebral hypoperfusion, ischemia and neuronal damage. Then, laparotomy with suturing of liver laceration can be carried out. The timing of surgery for non-life-threatening orthopedic surgery is controversial. Hypotension in the first 24–48 hours of head injury is associated with a worse outcome. If surgery is necessary within these hours, then it should proceed under ICP control with careful maintenance of CPP, oxygenation, PaCO₂, and avoidance of anemia.

Q. Enumerate advanced monitoring.

Ans. Advanced monitoring has been listed as intracranial pressure monitoring, jugular bulb venous oxygen saturation,

brain tissue oxygen tension, cerebral microdialysis, transcranial Doppler ultrasonography, electrophysiological monitoring, near-infrared spectroscopy, and brain temperature.

Q. How will you manage the patient of traumatic brain injury according to the severity of brain injury?

Ans.

Mild brain injury (GCS 13–15): Proper history taking and general examination to rule out systemic injuries and focused neurological examination. Investigations—X-ray cervical spine and other X-rays as indicated, blood alcohol levels, urine toxicology, CT scan if increased risk of neurosurgical intervention. Admission to an appropriate facility if no CT scan, or abnormal CT, skull fracture, CSF leak, focal neurological deficit or GCS does not return to 15 within 2 hours. Consider discharge if patient has no criteria for admission but emphasize need for follow-up.

Moderate brain injury (GCS 9–12): Initial examination, CT scan and trauma center admission, frequent neurological checks and repeat CT scan in case of deterioration or prior to discharge. If improvement is present, patient can be discharged after being advised for follow-up, if deterioration occurs, the patient is managed as per severe brain injury protocol after repeat CT scan (ATLS guidelines, 10th edition, page 170).

Severe brain injury (GCS <8): Initial ATLS scheme for management as mentioned above in the chapter. Therapeutic measures such as mannitol, hypertonic saline to decrease ICP, avoid hyperventilation for first 24 hours unless signs of herniation, with neurological reevaluation (GCS, pupillary examination), and CT scan.

Q. What are the physiological effects of raised ICP? Discuss management of intracranial hypertension (ICH).

Ans. The manifestations of physiological effects of raised ICP are hypertension, bradycardia, wide pulse pressure—Cushing's triad and ventricular dysrhythmias. Treat ICH with immediate interventions aimed at lowering ICP and increasing CPP with an aggressive intent as sustained ICP ≥ 20 mm Hg for > 10 minutes is associated with poor outcomes.

Airway management: GCS score ≤ 8 requires intubation. Therapeutic hyperventilation may be required to reduce ICP through vasoconstriction but should be done only in moderation and for a limited period. Maintain PaCO_2 at the lower end of normal range.

Maintain CPP: Reduce ICP and ensure adequate MAP to maintain CPP >60–70 mm Hg with fluid resuscitation and vasopressor support. Vasodilators such as diazoxide, hydralazine, etc. should not be used.

Maintain SBP at ≥ 100 mm Hg for patients (50–69 years) or at ≥ 110 mm Hg for patients 15–49 years or older than 70 years as it improves outcomes.

Osmotic diuresis: Mannitol (20%) is given as 0.5–1 g/kg bolus and repeated every 6–8 hours to maintain serum osmolality >310 mOsm/L. Avoid hypovolemia and hypotension as it reduces CPP. Use with ICP monitor, unless there is evidence of herniation, and use bolus rather than continuous infusion. Hypertonic saline is given to maintain sodium at upper limits of normal as hyponatremia indicates poor outcomes. It increases serum osmolality, which expands intravascular volume, improving CBF, and facilitates fluid removal in cerebral edema.

Avoid hyperthermia to prevent increase in ICP but hypothermia which reduces ICP may mediate apoptosis, coagulopathy, and electrolyte abnormalities. Shivering may increase ICP. No role of therapeutic hypothermia. *There is no role of steroids in the head injured patients.*

Glycemic control: Treat hyperglycemia aggressively as it leads to poor outcomes.

Postural drainage: 15–30° head elevation with neck maintained in midline position to facilitate venous return. Avoid jugular central venous lines and improperly positioned cervical collar as it obstructs venous drainage.

Sedation and analgesia: Avoid and treat pain and agitation as it increases ICP. Benzodiazepines and propofol increase seizure threshold, therefore beneficial. Excessive sedation and analgesia masks examination findings prolonging the course of mechanical ventilation.

Neuromuscular blockade: Therapeutic paralysis reduces ICP but masks seizure activity, negates the physical examination; potentially delaying early signs of deterioration, prolongs ventilation, and increases risk for pressure ulcers and venous thromboembolism. Deep sedation is safer while providing similar benefits.

Anticoagulants: Exercise caution in patients on anticoagulants. All supra-therapeutically anticoagulated patients and CT abnormality need to be admitted for observation and repeat CT at 12–18 hours or at signs of neurologic worsening.

Barbiturate coma: Considered in patients with refractory ICH or with uncontrolled seizures. Continuous bedside EEG monitoring is necessary to monitor adequate burst suppression with infusions.

Cerebrospinal fluid drainage through an intraventricular catheter to reduce ICP. Drainage should be continued to maintain the ICP between 5 and 15 mm Hg; however, increases chances of infection.

Decompression craniectomy: Decreases ICP and improve outcomes if other therapies are ineffective. Wide decompressive craniectomy (DC) with duroplasty is effective in decreasing raised ICP in intraparenchymal contusions.

Q. Describe surgical management for various lesions in head injured patient.

Ans.

Subdural hematoma (SDH) with thickness >10 mm or midline shift >5 mm on CT scan—evacuated irrespective of GCS. SDH <10 mm thick or midline shift <5 mm with GCS < 9 can undergo surgery if GCS decreases by two points from admission and presents with asymmetric or fixed pupils, or if ICP > 20 mm Hg.

Extradural hematomas: Epidural hematomas (EDH) (>30 cm³)—evacuated irrespective of GCS status. An EDH < 30 cm³ and thickness < 15 mm with <5 mm midline shift with GCS score > 8; without neurological deficit—conservative management with CT scanning and neurologic monitoring.

Traumatic parenchymal lesions with signs of neurological deterioration, medically refractory raised ICP, signs of mass effect on CT scan—treat operatively. Frontal and temporal contusions >20 cm³ with GCS score of 6–8 and midline shift of at least 5 mm and cisternal compression on CT scan and lesions >50 cm³ should be operated.

Posterior fossa lesions with mass effect on CT scan or neurological deterioration require urgent surgery (suboccipital craniectomy for hematoma evacuation) as they are likely to deteriorate rapidly.

Q. Discuss postoperative care of a head injured patient. How would you give prophylaxis against DVT? Should this patient be given antiepileptic and for how long?

Ans. If the patient has undergone only an evacuation of extradural hematoma, he can be extubated once wide awake. If additional injuries such as diffuse axonal injuries present, then ventilation for a longer period may be required. Peak brain edema can take 24–72 hours to occur. Postoperatively, aim to prevent secondary injuries due to physiological factors causing cerebral edema, observe for any deterioration, and provide adequate analgesia. Any further insult can be detected by changes in level of consciousness, development of focal neurodeficit or unequal pupils. Mechanical prophylaxis using graduated stockings or sequential calf compression devices is used in the postoperative period. Avoid use of unfractionated heparin or low-molecular-weight heparin (LMWH) to prevent intracranial hematoma expansion. Prophylactic treatment with phenytoin or carbamazepine should be initiated early after injury to decrease the risk of post-

traumatic seizures and then stopped after 7 days. Not recommended for late seizures.

Summary for Traumatic Brain Injury [as per the Brain Trauma Recommendations, 4th Edition (2016)]

- Maintain SBP $\geq$ 100 mm Hg for patients (50–69 years) or $\geq$ 110 mm Hg for 15–49 years or over 70 years.
- Avoid PaO₂ < 60 mm Hg and SaO₂ < 90%, treat ICP > 20–25 mm Hg. Consider ICP monitoring appropriate with severe head injury and CT abnormalities. Maintain CPP at 60–70 mm Hg.
- No role of prolonged prophylactic hyperventilation with PaCO₂ of 25 mm Hg or less. No role of steroids.
- Boluses of mannitol are better than infusion for controlling ICP. Aim to keep serum osmolality < 320 mOsm.
- High-dose barbiturate administration and propofol infusion for elevated refractory ICP. Anti-seizure prophylaxis only for early seizures, no role for late seizures. Temperature should be maintained at 36–38°C. There is no role of prophylactic hypothermia.
- Feeding patients to attain basal caloric replacement at least by the fifth day and, at the most, by the seventh day postinjury.
- Decompressive craniectomy—a large frontotemporoparietal DC improves neurologic outcomes in patients with severe traumatic brain injury (TBI).
- Cerebrospinal fluid drainage—external ventricular drain (EVD) system zeroed at midbrain with continuous drainage more effective in lowering ICP than intermittent use.
- Infection prophylaxis—early tracheostomy is recommended to reduce mechanical ventilation days when the overall benefit is felt to outweigh the complications. Use of povidone-iodine oral care is not recommended to reduce ventilator-associated pneumonia and may increase risk of acute respiratory distress syndrome (ARDS). Antimicrobial-impregnated catheters may be considered to prevent catheter-related infections during EVD.
- Advanced cerebral monitoring—jugular bulb monitoring of arteriovenous oxygen content difference with jugular venous saturation of < 50% as a threshold to improve outcomes and reduce mortality.

Q. Highlight the updates in pediatric brain trauma guidelines (2019 updates in pediatric traumatic brain injury guidelines).

Ans.

- Intracranial pressure monitoring is recommended improving overall outcome.
- Advanced neuromonitoring to improve overall outcomes. If brain tissue oxygenation (PbrO₂) monitoring is used, maintaining level > 10 mm Hg is suggested.

- Threshold for CPP minimum of 40 mm Hg and treatment of ICH < 20 mm Hg is suggested.
- *Hyperosmolar therapy*: Bolus hypertonic saline (3%) is recommended in patients with ICH. Recommended effective doses range between 2 and 5 mL/kg over 10–20 min, continuous infusion of 3% saline ranges between 0.1 and 1.0 mL/kg of body weight per hour. Bolus of 23.4% hypertonic saline is suggested for refractory ICP. The suggested dose is 0.5 mL/kg with a maximum of 30 mL.
- Seizure prophylaxis to reduce incidence of early (within 7 days) post-traumatic seizures. Insufficient evidence to recommend levetiracetam over phenytoin based on either efficacy or toxicity.
- Decompressive craniectomy for neurologic deterioration, herniation, or refractory ICH.
- *High-dose barbiturate therapy*: In hemodynamically stable patients with refractory ICH.
- Corticosteroids *are not suggested* to improve outcome or reduce ICP.

Recommendations regarding nutrition, ventilator strategies, CSF drainage, prophylactic hypothermia, are similar to adults.

■ ACKNOWLEDGMENT

We acknowledge the help of Dr Ashwini Wanjary, Junior Resident, Department of Anesthesiology, Critical Care and Pain for her artistic contribution to the chapter for drawing Figure 1.

■ SUGGESTED READING

1. Adamides AA, Winter CD, Lewis PM, Cooper DJ, Kossmann T, Rosenfeld JV. Current controversies in the management

- of patients with severe traumatic brain injury. *ANZ J Surg.* 2006;76:163-74.
2. Bor-Seng-Shu E, Figueiredo EG, Amorim RL, Teixeira MJ, Valbuza JS, de Oliveira MM, et al. Decompressive craniectomy: a meta-analysis of influences on intracranial pressure and cerebral perfusion pressure in the treatment of traumatic brain injury. *J Neurosurg.* 2012;117:589-96.
3. Edwards P, Arango M, Balica L, Cottingham R, El-Sayed H, Farrell B, et al. Final results of MRC CRASH, a randomised placebo-controlled trial of intravenous corticosteroid in adults with head injury-outcomes at 6 months. *Lancet.* 2005; 365:1957-9.
4. Eken C, Kartal M, Bacanlı A, Eray O. Comparison of the Full Outline of Unresponsiveness Score Coma Scale and the Glasgow Coma Scale in an emergency setting population. *Eur J Emerg Med.* 2009;16:29-36.
5. Guidelines for the management of severe traumatic brain injury. *J Neurotrauma.* 2007;24:S1-106.
6. Haddad SH, Arabi YM. Critical care management of severe traumatic brain injury in adults. *Scand J Trauma Resusc Emerg Med.* 2012;20:12.
7. Ramani R. Hypothermia for brain protection and resuscitation. *Curr Opin Anesthesiol.* 2006;19:487-91.
8. Rosenfeld JV, Maas AI, Bragge P, Morganti-Kossmann MC, Manley GT, Gruen RL. Early management of severe traumatic brain injury. *Lancet.* 2012;380:1088-98.
9. Skoglund TS, Eriksson-Ritzén C, Jensen C, Rydenhag B. Aspects on decompressive craniectomy in patients with traumatic head injuries. *J Neurotrauma.* 2006;23:1502-9.
10. Urbano LA, Oddo M. Therapeutic hypothermia for traumatic brain injury. *Curr Neurol Neurosci Rep.* 2012;12:580-91.
11. Wakai A, McCabe A, Roberts I, Schierhout G. Mannitol for acute traumatic brain injury. *Cochrane Database Syst Rev.* 2013;(8):CD001049.
12. Xiong Y, Mahmood A, Chopp M. Emerging treatments for traumatic brain injury. *Expert Opin Emerg Drugs.* 2009;14: 67-84.

Managing Difficult Airway

Vijaya Prakash Patil, Jeson Rajan Doctor, Debashree Lahiri

Induction of general anesthesia (GA) followed by direct laryngoscopy and oral intubation can be difficult, if not impossible, in several situations. To determine the optimal intubation technique, the anesthesiologist must elicit an airway history and carefully examine the patient's head and neck. Any available prior anesthesia records should be reviewed for previous problems in airway management. If a facial deformity is severe enough to preclude a good mask seal, positive-pressure ventilation may be impossible. Furthermore, patients with hypopharyngeal disease are more dependent on muscle tone (while awake) to maintain airway patency. These two groups of patients should not be allowed to become apneic for any reason—including induction of anesthesia, sedation, or muscle paralysis—until their airway is secured. In other patients in spite of thorough airway evaluation one has to remember surprises can crop up and anesthesiologist should be ready with options to manage difficult airway at any time. In short, easy to intubate on history taking and clinical examination does not necessarily mean easy intubation.

Q. What are some of the common screening tests employed to assess the airway?

Ans. Visual inspection and examination: Face, teeth, incisors, nose, palate, tongue, mandible, tonsils, oral cavity, and neck.

Single Parameter Tests

- *Assessment of temporomandibular joint (TMJ) function:*
 - *Interincisor gap/mouth opening:* >5 cm or normally >3 fingers
 - Jaw protrusion
 - *Upper lip bite test:*
 - Class I lower incisors can bite upper lip above the vermilion line
 - Class II lower incisors can bite upper lip below vermilion line
 - Class III lower incisors cannot bite upper lip.
 - *Movement of condyle of mandible in front of the tragus.*
- *Assessment of mandibular space and floor of mouth for tongue displacement:*
 - *Thyromental distance:* Normal > 6.5 cm (four fingers)
 - *Hyomental distance:* Normal > 6 cm (three fingers)
 - *Mandibular angle to mental symphysis distance or horizontal length of mandible:* At least 9 cm.
- *Assessment of adequacy of the oropharynx for laryngoscopy and intubation:*
 - *Mallampati classification (Samsoon and Young modification):* Pharynx to tongue ratio. Examiner sits opposite to the patient at eye level and the patient opens his mouth as wide as possible and protrudes the tongue without phonating:
 - *Grade 1:* Soft palate, uvula, tonsillar fauces, and posterior pharyngeal wall visible
 - *Grade 2:* Soft palate, uvula, and tonsillar fauces visible
 - *Grade 3:* Soft palate and base of uvula seen
 - *Grade 4:* Only hard palate visualized
 - *Grade 0:* Epiglottis seen.
 - *Assessment of cervical and atlanto-occipital (AO) joint function:* Optimal position for direct laryngoscopy involves flexion of cervical spine and extension of AO joint (Sniffing position aligns oropharyngeal and laryngeal axes to a favorable line of sight). If the patient can touch his manubrium sterni with his chin this assures neck flexion of 25–30°. The patient is now asked to open his mouth wide such that the occlusal surface of the upper incisor teeth is parallel to the ground. On extending the AO joint, the angle created by movement of the occlusal surfaces is estimated. It should be at least 80–85°.
 - *Thyroid to floor of mouth distance:* It indicates the position of the larynx in the neck. It is normally placed

if the patient can place two fingers between the top of the thyroid cartilage and the floor of the mouth.

- **Sternomental distance:** This is measured with the head in full extension and the mouth closed. Normal distance is >12.5 cm.
- **Quality of glottic view during laryngoscopy:** Not usually done in an OPD setting. Useful in cases of recorded previous difficult intubation:
 - Indirect mirror laryngoscopy or Hopkin's examination.
 - **Direct laryngoscopy:** Done under topical anesthesia (awake look) or with the patient under inhalational or intravenous (IV) induction but not apneic or paralyzed or patient completely under anesthesia and paralyzed. Cook's modification of Cormack and Lehane's grading:
 - **Grade I:** Visualization of entire vocal cords
 - **Grade IIa:** Visualization of posterior part of vocal cords
 - **Grade IIb:** Visualization of arytenoids only
 - **Grade IIIa:** Epiglottis liftable
 - **Grade IIIb:** Epiglottis adherent or only tip visible
 - **Grade IV:** No glottic structures seen
 - **POGO scoring:** Discussed in detail later.

Group Indices: Multiple Parameter Tests

- **Wilson's scoring system:** Based on weight of patient, head and neck movement, interincisor gap presence of buck teeth and receding mandible. It has a total score of 10. Score <5 = Easy laryngoscopy, 6–7 moderate difficulty, 8–10 severe difficulty.
- **Benumof's 11 parameter analysis (first four parameters for teeth):** Length of upper incisors, interincisor gap, involuntary buck teeth override, voluntary protrusion of mandibular teeth in relation to maxillary teeth (next two parameters for intraoral structures), MPC, palatal configuration and narrowness (next two for mandibular space), thyromental distance, compliance of mandibular space (last three parameters for neck), neck length, neck thickness, and head and neck movement.

Radiological Assessment

- X-ray neck cervical spine (anteroposterior/lateral):
 - Atlanto-occipital gap <5 mm
 - C1-C2 gap <5 mm
 - Airway compression/deviation
 - Mandibular length/depth ratio.
- CT scan—if available
- MRI—if available.

Advanced Assessment Tests

- Flow volume loops—if available
- Acoustic response measurement and USG.

Assessment for Difficult Bag Mask Ventilation

BONES

- Bearded individuals
- Obese individuals
- No teeth
- Elderly
- Snorers.

Assessment for Difficult Supraglottic Airway Device Placement

RODS

- Restricted mouth opening
- Obstructed upper airway
- Disrupted upper airway following trauma and burns
- Stiff lungs (poor lung or thoracic compliance).

Assessment for Difficult Surgical Airway

BANG

- Bleeding tendency
- Agitated patient
- Neck scarring or flexion deformity
- Growth or vascular abnormalities in the region of the surgical airway.

In addition to the above-mentioned, if the patient is to be posted for emergency surgery the risk of aspiration along with a difficult airway should also be borne in mind.

Q. Why do we get unanticipated difficult intubations? Why do predictive tests fail?

Ans. Four terms provide the information needed to analyze the usefulness of a predictive test.

Sensitivity: Test sensitivity is a measure of whether it identifies correctly the difficult patients as being difficult. A test sensitivity of 80% indicates that 80% of the difficult patients will be identified correctly as difficult, and 20% will be missed and classified as not-difficult or normal. A test sensitivity of 100% is ideal.

Specificity: Specificity of a test identifies that a normal patient is normal. A specificity of 80% indicates that 80% of normal patients will be correctly identified as normal, but 20% of normal patients will be identified incorrectly as difficult. A test specificity of 100% is ideal.

Positive predictive value (PPV): The positive predictive value is the percentage that is true difficult intubations out of all those predicted by the test to be difficult.

Likelihood ratio: The likelihood ratio (LR) is an extremely useful term and can be calculated within seconds using only the sensitivity and specificity. The LR is the chance of a positive test if the person is difficult, divided by the chance of a positive test if the patient is normal.

From **Table 1**, it is clear why prediction tests fail.

TABLE 1: Predictive ability of common tests.

Test	Sensitivity (%)	Specificity (%)	PPV (%)
Mallampati (original)	42–60	81–89	4–21
Mallampati (modified)	65–81	66–82	8–9
Thyromental	65–91	81–82	8–15
Sternomental	82	89	27
Wilson score	42–55	86–92	6–9
Mouth opening	26–47	94–95	7–25
Jaw protrusion	17–26	95–96	5–21

(PPV: positive predictive value).

Q. Enumerate basic principles of difficult airway management.

Ans.

- *Preparation of team (anesthetists, surgeon, nurse, technician/OT attendant):* Equipment, right frame of mind, well-defined primary and back-up plans.
- Good airway preparation with antisialogogues and local anesthetics.
- Obtaining maximum cooperation from patient.
- Maintenance of spontaneous breathing till airway is secured.

Q. What is intubation difficulty scale?

Ans. The intubation difficulty scale (IDS) was proposed in 1997 and incorporates seven variables (given below) to describe the ease or difficulty of a particular intubation by direct laryngoscopy. The sum of each variable produces the IDS score allowing a gradation of intubation from easy through to impossible, rather than a simple difficult/not difficult definition. The first three (N1-3) variables have no upper limit; the fourth variable (N4) is glottic exposure according to the Cormack and Lehane four grades minus one. A Grade 1 view is awarded zero points and a Grade 4 view three points. Successful blind nasotracheal intubation scores 0. The glottic exposure is evaluated during the first attempt by the first operator. The last three variables (N5-7) are scored either 0 or 1. An IDS of 0 indicates intubation without difficulty and there is no upper limit. The cutoff to define difficult intubation is arbitrary but a value of five has been used in more than one study. In a prospective study of 1,171 patients an IDS of 0 was found in 55% patients and IDS >5 occurring in 8% patients. It may be very useful in communicating the total intubating difficulty for a given patient to the next caregiver. It should be noted that a particular score may be obtained through differing problems and any score, on its own, is not diagnostic. It is ideal to transmit information in case of difficult/failed intubation and is only of practical use if the score for each of the seven variables is recorded and transmitted to the next anesthetist.

- N1: Number of attempts >1
- N2: Number of operators >1
- N3: Number of alternative techniques
- N4: Cormack-Lehane grade-1
- N5: Lifting force required (normal 0 or increased 1)
- N6: Laryngeal pressure (not applied 0 or applied 1)
- N7: Vocal cord mobility (abduction 0 or adduction 1).

Q. What are predictors of difficult mask ventilation?

Ans.

Predictors include:

- BMI >30 kg/m²
- Beard
- Age >57 years
- Snoring
- Limited jaw protrusion
- Abnormal neck anatomy
- Sleep apnea
- Thyromental distance <6 cm
- **O:** Obese
- **B:** Beard
- **E:** Elderly
- **S:** Sleep apnea, secretions
- **E:** Edentulous.

Q. What is percentage of glottic opening (POGO) score?

Ans. The percentage of glottic opening (POGO) score represents the portion of the glottis visualized. Glottis is defined anteriorly by the anterior commissure and posteriorly by the interarytenoid notch. The score ranges from 0% when none of the glottis is seen to 100% when the entire glottis including the anterior commissure is seen. The POGO score does not differentiate between visualization of the epiglottis and the tongue. In each case, the POGO score would be 0. The POGO score (**Fig. 1**) is a simple and an easy way to categorize laryngeal view. Studies have found that it has better interphysician reliability than Cormack-Lehane grading.

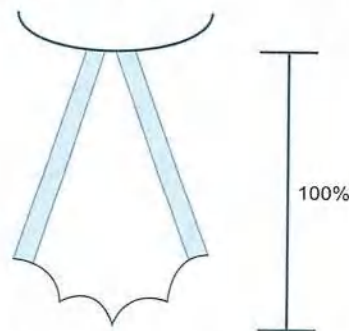


Fig. 1: Percentage of glottic opening (POGO) score.

Q. What is ramp position and where is it useful?

Ans. It is also called as head-elevated laryngoscopy position (HELP) position. If the patient is obese, it may be necessary

to make a “ramp” of pillows or blankets under her head, shoulders and upper back to achieve the proper airway angle and a sniffing position. Dr Richard Levitan, inventor of the Airway Cam, calls this the HELP, originally introduced by Chevalier Jackson at the beginning of the 20th century. The shoulders and head should be raised enough so that an imaginary horizontal line drawn through the ear canal intersects the xiphoid process. Rescue ventilation techniques (oral airway/bag and mask) are facilitated by the HELP position. Since head and neck are elevated above the

chest and abdomen, the airway is more isolated and easier to work with. Further, the weight of the abdomen falls away from the diaphragm allowing better ventilation. Positioning the morbidly obese patient has shown that this position improves the laryngoscopy view and also eases ventilation and improves oxygenation allowing more apneic period. Stacking with blankets can create the HELP position, but this may cause head and neck to be unstable. A pre-cut foam positioner designed to quickly achieve the HELP position is commercially available and preferred (**Figs. 2 and 3**).

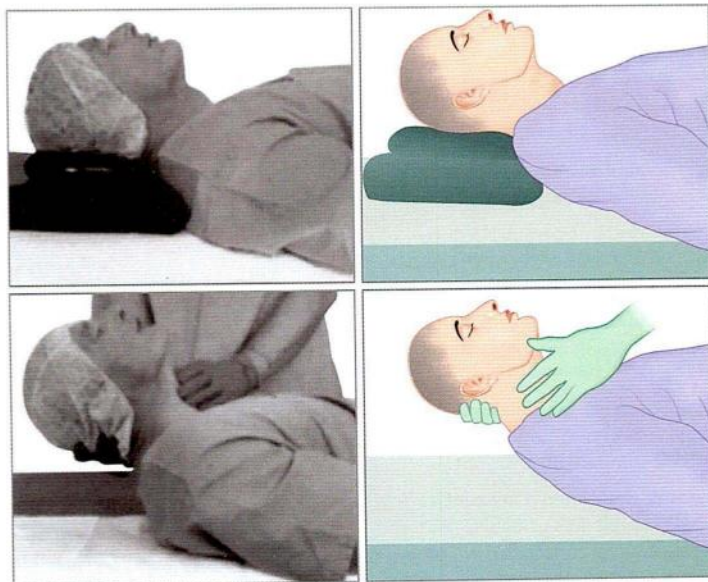


Fig. 2: Ramp position.

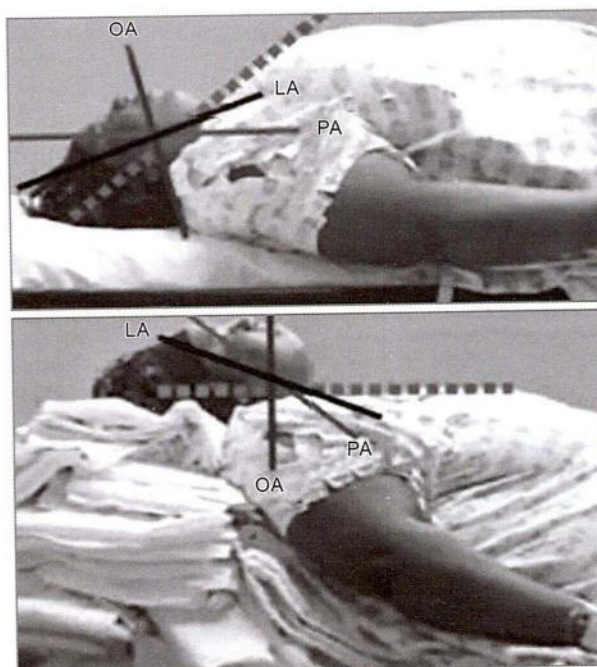


Fig. 3: Alignment of airway axes. (LA: laryngeal axis; OA: oral axis; PA: pharyngeal axis).

Q. What are the contents of airway cart/difficult intubation trolley?

Ans. Anesthetists should be ready to deal with difficulties in airway management at any time. The correct equipment must be immediately available. It is not only important to have all equipment but equally important to get correct equipment in emergency without need to search through pile of gadgets. A systematic approach of stocking these various gadgets helps in saving time. Difficult Airway Society (DAS) has suggested having a cart with various drawers with designated equipment in each drawer. The contents of the difficult airway cart are as follows (**Table 2 and Fig. 4A**).

TABLE 2: Contents of difficult airway trolley.

Top of trolley	Flexible fiber-optic bronchoscope (FOB)
Side of trolley	Bougies (adult/pediatrics), Aintree intubation catheter, airway exchange catheter
Drawer 1 (plan A)	Bougies, short handle laryngoscope, McCoy blade and/or straight blade, videolaryngoscope
Drawer 2 (plan B)	LMA or second-generation SAD (3, 4, 5), ILMA, Aintree intubating catheter, fiber-optic adjuncts (such as oral airway, nasal sponge, 4% lignocaine)
Drawer 3 (plan C)	Face masks (various sizes), oropharyngeal airways (various sizes), nasopharyngeal airways (various sizes), LMA/Proseal (3, 4, 5)
Drawer 4 (plan D)	Large bore cannula device, e.g., Quicktrach I or II (VBM), scalpel (No. 10 blade), tracheal dilator or tracheal hook, bougie, cuffed tracheal tube #6 and 7, kink-resistant jet ventilation cannula, e.g., Ravussin (VBM), high and/or low pressure ventilation system, e.g., Manujet III (VBM) Ventrain (Ventinova)

(ILMA: intubating laryngeal mask airway; LMA: laryngeal mask airway; SAD: supraglottic airway device).

**Fig. 4A:** Difficult airway cart.**Q. Enumerate the various systemic diseases which are known to make the airway difficult.**

Ans.

Systemic disease	Airway involvement
Diabetes mellitus	- Limited joint mobility syndrome - Risk of difficult airway is associated with age, diabetes duration, and degree of glycemic control - Limited atlanto-occipital joint motion - No relation between the prayer sign and difficult laryngoscopy - Palm print test most sensitive and specific for difficult laryngoscopy
Rheumatoid arthritis	- Mainly affects cervical spine - Atlantoaxial instability or subluxation is the most common abnormality - Superior migration of the odontoid is the next most common - Least common deformity is subaxial subluxation - Involvement of cricoarytenoid joint—impaired mobility or fixation - Swelling of the arytenoids, aryepiglottic folds and epiglottis - TMJ may be involved causing limited MO
Acromegaly	- Prognathism - Macroglossia - Thickening of pharyngeal and laryngeal soft tissue and vocal cords - Fixation of vocal cords - Recurrent laryngeal nerve palsy - Decreased width of cricoid arch - Hypertrophy of aryepiglottic and ventricular folds 70% patients have OSA
Cushing's	- Difficult airway is due to obesity and OSA "Buffalo hump" may interfere with supine positioning and impede airway management
Hypothyroidism	- Myxedema of upper airway—rare - Hypertrophic myopathy of pharyngeal dilator muscles—rare
Obesity	- Excess soft tissue around neck and in oropharynx—difficult mask ventilation - Difficult laryngoscopy and intubation - Increased risk of regurgitation and aspiration - BMI of ≥ 35 predicts difficult tracheal intubation with a sensitivity of 7.5% and a PPV 6.4%
Trauma	- Cervical spine injury - Maxillofacial fracture - Laryngotracheal fractures - Foreign body aspirations
Burns	- Physiologically difficult airway - Edema of injured and noninjured tissues
Anaphylaxis	- Airway edema - Bronchospasm

Contd...

Contd...

Systemic disease	Airway involvement
Amyloidosis	• Macroglossia • Laryngeal amyloid • Esophageal dysmotility needing RSI
Sarcoidosis	• Endobronchial disease is common • Laryngeal/pharyngeal involvement • Erythematous nodules on turbinates and septum
Wegener's granulomatosis	• Frequency of upper airway involvement during disease course is 92% and lower airway involvement is 85% • Subglottic stenosis is the most characteristic and serious laryngeal lesion—common in children
Diphtheria/Croup	• Laryngeal diphtheria leads to characteristic "bull neck" • Pharyngeal edema, leathery membrane variably covers the tonsils, soft palate, oropharynx, nasopharynx, and uvula

(BMI: body mass index; TMJ: temporomandibular joint; OSA: obstructive sleep apnea; PPV: positive predictive value; RSI: reflux symptom index).

Q. What are National Audit Projects (NAPs) and what is special about fourth National Audit Project (NAP4)?

Ans. The National Audit Projects study anesthesia-related complications of low incidence that are potentially serious for patients and important to patients and anesthetists. NAP4 was a joint project of Royal College of Anesthetists and the Difficult Airway Society to study the incidence of major complications of airway management during anesthesia in UK. The audit suggested that majority of the events and deaths reported were likely to be avoidable. However, the incidence of serious complications associated with anesthesia is low. The same holds true for airway management in intensive care unit (ICU) and the emergency department, though it is likely that a disproportionate number of airway events occur in these locations. The aim of this report was to make airway management safer. The important findings from this report are:

- When difficult airways were encountered, there was often no documentation of their having previously been sought; yet where there was documentation of difficult airway assessment, the difficult airway was usually identified and therefore planned for.
- About 29% of adverse events occurred at the end of the procedure and were most commonly a result of airway obstruction.
- Gastric content aspiration was the major contributor to airway-related morbidity and mortality, rather than procedural hypoxia.
- Needle cricothyroidotomy as an airway rescue procedure had a 75% failure rate, prompting consideration of a lean towards a surgical cricothyroidotomy as the preferred airway rescue procedure.

- In ICU airway events contributed 20% of total adverse events but >50% of mortality and >60% of death or brain damage.
- Incidence of airway related events were greater during out of hours. Procedural hypoxia was the primary contributor to adverse outcomes. Airway events were largely related to unfamiliarity with difficult airway assessment, difficult airway procedures and equipment and dislodgement of tracheostomies.
- The leading human factors contributing to a serious adverse outcome that were identified were: poor communication, poor teamwork, poor leadership and task fixation.

■ CASE SCENARIOS

Case 1: A 32-year-old, 58 kg male patient diagnosed to have ankylosing spondylitis was referred for the spine surgery. He gives history of gradual onset, progressive painless deformity since 4 years, resulting in thoracolumbar kyphosis. He cannot look up or straighten himself while walking. He cannot lie down supine, and needs 2–3 pillows, beneath his head to support it while sleeping.

Ankylosing spondylitis leads to fibrosis, ossification, and ankylosis along the spinal column and sacroiliac articulations. Cervical column and AO articulation mobility are reduced and in severe cases the cervical vertebrae become fixed in a flexed position. This portion of the spine is also the most susceptible to fracture, particularly in hyperextension, and can damage the cervical spinal cord during maneuvers to manage the airway. Patients with this condition may also have TMJ involvement, further complicating airway management. Preferably they should be managed along the awake limb of difficult airway algorithm.

Q. How will you prepare a patient for awake intubation? What premedication will you use?

Ans. Adequate preparation is must for success. An antisialogogue, administered in sufficient time to work, is imperative. IV scopolamine, atropine or glycopyrrolate may be used. We routinely use glycopyrrolate because it is nonsedative and results in fewer hemodynamic and central side-effects than atropine. The nasal passage (if the nasal route is chosen) and the oropharynx, cords, and trachea should be anesthetized with local anesthetic agent, the most commonly used agent being lignocaine. The appropriate level of analgesia and sedation is challenging because overdose of medication resulting in the loss of spontaneous ventilation can be disastrous in a difficult airway, and insufficient medication may result in patient discomfort, pain, coughing, and tachycardia, making the tracheal intubation technically more difficult to even impossible. Use

titrated amount of short-acting sedatives and opioids such as midazolam, dexmedetomidine, and fentanyl to a Ramsay scale 2–3. Adequate topical anesthesia in conjunction with light sedation usually produces very satisfactory intubating conditions. The nasal passage should be anesthetized with 2% lignocaine jelly in case nasal route is selected. Vasoconstriction of nasal mucosa is very important and can be accomplished by using phenylephrine/adrenaline soaked pellets or nasal decongestants like metazolines.

Q. How would you anesthetize the airway for awake intubation?

Ans. Translaryngeal block: Placement of local anesthetic within the larynx can provide anesthesia of the larynx and trachea below the vocal cords. Sensory supply to larynx below vocal cords is by the recurrent laryngeal nerve, and above vocal cords is by superior laryngeal nerve both being branches of vagus nerve. This block should be avoided when patient coughing is undesirable [e.g., raised intracranial pressure/intraocular pressure (ICP/IOP)].

To perform the block, identify the cricothyroid membrane, which can be very easily identified by running the finger in the midline of neck in extension. Cricothyroid membrane will be felt as a soft depression just below Adams apple. Place a 22-gauge needle through this membrane (using smaller gauge needle prevents rapid injection of LA in trachea. Needle can get displaced with coughing of patient when LA is being injected slowly and prevent instillation of full volume of LA in trachea when finer needles are used. This may lead to unsatisfactory anesthesia). Feeling of sudden give way marks entry into trachea. Aspirate air from the trachea to confirm the position. Inject 3–5 mL of local anesthetic (4% lignocaine) rapidly and withdraw the needle. The patient will immediately cough and spread the local anesthetic up and down the larynx and trachea.

Superior laryngeal nerve block: The superior laryngeal nerve is a branch of the vagus nerve. It travels just inferior to the greater cornu of the hyoid bone and divides into internal and external branches to provide sensation to the anterior and posterior epiglottis and laryngeal mucosa to the level of the vocal cords.

To perform the block, gently displace the hyoid bone toward the side to be blocked. Place a 24/25-gauge needle just inferior to the greater cornu of the hyoid bone and superior to the thyroid cartilage and inject 2–3 mL of local anesthetic. 1% lignocaine may be used. Repeat the procedure on the other side.

Nebulization: Same can be achieved by using nebulization with 4% lignocaine before procedure.

While using all these techniques one must take care to calculate total dose of LA used and restrict amount to safe maximum dose (3–5 mg/kg for lignocaine).

Q. What are the different ways of securing the airway in an awake patient?

Ans. Blind nasal intubation: This technique is not entirely blind as name suggests but it is guided by breath sounds. Here a nasal endotracheal tube is gently passed through the nose toward the larynx. One should continuously hear breath sounds and guide the tube in the direction of the loudest breath sounds by moving the patient's head until the larynx is entered. This technique can be employed for patients with trismus where mouth opening is limited but the oropharyngeal and laryngeal anatomy is normal. This technique requires a great deal of skill and expertise of operator and cooperation of patient and is not feasible if the head and neck cannot be moved.

Retrograde intubation is a technique first described in Nigeria for intubation of patients with cancrum oris. A soft wire or epidural catheter is passed through the cricothyroid or cricotracheal membrane in a cephalad direction (toward the head) until it comes out of the nose or mouth. (In some patients it is necessary to grasp the catheter in the mouth using a pair of Magills forceps). An endotracheal tube is then inserted over this wire into the trachea from either the nasal or oral route. Ensure oxygenation is maintained throughout. During the technique, if a nasal intubation is required but the wire comes out of the mouth, place another catheter through the nose, pull it out through the mouth, tie it to the wire, and then pull the wire up through the nose. Due to discrepancy between thin wire/catheter and large endotracheal tube many times it becomes difficult to guide tube into trachea. To overcome this problem, some researchers advocate doing this in two stages—use of bougie or airway exchange catheter over wire, and then guiding tube over bougie or airway exchange catheter.

Fiber-optic intubation: Fiber-optic intubation is simple in principle, but requires practice to learn. If one has to master the technique one has to use the scope frequently. Familiarity with the scope features and practice in manipulation should be obtained before clinical use to decrease damages of costly equipment. Tip control is quite simple, up/down motion is adjusted with the lever and side-to-side motion is accomplished by clockwise/counter-clockwise rotation of the entire scope-handle and shaft. Trying to achieve this rotation by turning the handle alone (without simultaneous rotation of the shaft with the other hand) produces torque which can damage the scopes especially if it is passing through a snug fitting endotracheal tube and should be avoided at any cost as this twisting is one of the most common causes of scope damage. Nasal approach is usually the preferred route compared to the oral one, as it is easier to navigate the scope.

The light wand: This consists of a handle, a wand and a stylet. On the handle, a locking clamp accepts and secures a standard endotracheal tube (ETT) connector. The distal end of the wand has a bright light bulb. To accommodate ETT of different lengths, the length of the wand can be adjusted by sliding the connector along the handle. The stiff, retractable stylet can be used to shape wand in a “hockey stick” shape. ETT-light wand and stylet are used together as one unit. When the tip of the ETT is in the glottic opening, a hockey stick bend allows the maximum light intensity to point to the surface of the skin. In a sniffing position, the epiglottis is almost in contact with the posterior pharyngeal wall, making it difficult for this assembly to pass through vocal cords. Therefore, it is recommended that the patient’s head and neck should be placed in a neutral or relatively extended position. In patients where neck extension cannot be given, the epiglottis can be lifted off the posterior pharyngeal wall with a simple jaw lift maneuver.

Laryngeal mask airway/intubating laryngeal mask airway: Introduced in 1981, it is a supraglottic device that shifts the mask seal from the face to a supraglottic location. There is plenty of literature describing successful insertion and use of this device by even nonmedical personnel. Intubating laryngeal mask airway (ILMA) was developed from MRI studies in nonobese patients for airway management, and it is easier to insert because of its curvature and handle and can be manipulated to achieve optimum seal.

Reported success rate of blind tracheal intubation via ILMA varies between 89.51 and 100%. Various methods used for intubation through ILMA are:

- Specialized flexometallic tubes that are provided with ILMA.
- Standard short bevel flexometallic tubes (armored tubes).
- Standard polyvinylchloride (PVC) endotracheal tubes (these are ideally not recommended but in the absence of ILMA tubes one size smaller PVC-ETT tubes may be used).
- Gum elastic bougie through ILMA.
- Airway exchanger—Cook or Patils.
- Fiber-optic bronchoscope through ILMA.

Awake tracheostomy performed under local anesthesia is the best solution when a patient is impossible to intubate, and regional anesthesia is not a practical option. This is a straightforward technique, except in children, when sedation with ketamine can be used to facilitate this approach.

Q. Discuss the salient features of the DAS guidelines for awake fiber-optic intubation.

Ans. In 2019, the DAS has released guidelines for awake tracheal intubation in adults in order to support anesthesia providers in decision making, preparation and practical

performance of awake tracheal intubation in adults. These guidelines aim to support clinical practice and thus lower the threshold for performing awake tracheal intubation when indicated. They recommend the following:

- Awake tracheal intubation must be considered in the presence of predictors of difficult airway management.
- A checklist is recommended before and during performance of awake tracheal intubation.
- Supplemental oxygen should always be administered during awake tracheal intubation. High-flow nasal cannula (HFNC) may be used for providing humidified oxygen.
- Effective topical anesthesia must be established and tested. The maximum dose of lidocaine should not exceed 9 mg/kg of lean body weight. This includes nebulization’s, oral sprays, spray as you go, atomizer device, transtracheal injections and IV preparations.
- Cautious use of minimal sedation with monitoring can be beneficial. This should however be administered by an independent practitioner. Short-acting agents such as dexmedetomidine and midazolam are preferred over propofol. Sedation is however not a substitute for good topicalization of the airway and should be used judiciously so that the airway is not lost.
- The number of attempts should be limited to three, with one further attempt by a more experienced operator (3 + 1).
- Subsequent attempts should change the operator/endotracheal tube/route and device.
- Anesthesia should only be induced after a two-point check (visual confirmation of endotracheal tube in trachea and capnography) has confirmed correct tracheal tube position.
- All departments should support anesthetists to attain competency and maintain skills in awake tracheal intubation.

The STOP approach includes **S**edation, **T**opicalization, **O**xygenation, and **P**erformance of the Procedure.

Case 2: A 17-year-old girl presents for emergency drainage of a submandibular abscess. How will you manage her airway?

Induction of GA followed by direct laryngoscopy and oral intubation is dangerous in these situations. To determine the optimal intubation technique, elicit a proper history and carefully examine patients head and neck. If hypopharynx is involved to the level of hyoid bone (evident from edema and redness), intubation will be very difficult. Also look for signs of airway obstruction such as stridor/chest retraction and signs of hypoxia such as agitation, restlessness, anxiety, lethargy, etc. Aspiration pneumonia is likely if the patient has eaten recently or if pus is draining in mouth and in both the cases abolition of reflexes should be avoided.

In the above case, on physical examination, there is extensive facial edema, with limited mouth opening due to pain, and frank pus is observed in mouth.

In this case, awake intubation is the only option. The blind nasal approach to awake intubation is a relative contraindication here, as this patient will have extensive intraoral edema, a distorted anatomy that can lead to more trauma and worsen situation further. Thus FOB-guided intubation is the technique of choice.

If the patient has adequate NBM status and there is no rupture of abscess cavity intraorally, one can try anesthetizing the patient maintaining spontaneous breathing and performing direct laryngoscopy with an attempt to visualize glottis and intubation. Regardless of technique, one has to be ready for a tracheostomy with an experienced surgeon in OT. Give antisialogogues, such as glycopyrrolate injection as a premedication to decrease secretions. Sedative premedication should be avoided if possible or given very carefully in titrated doses. Explain the need for awake intubation with stepwise approach to allay patients' anxiety and secure better cooperation.

Case 3: A 4-year-old malnourished child presents with huge adamantinoma for resection and reconstruction. On airway examination, we find mouth opening of 1.5 cm. Mallampati class II, normal neck movements.

Anesthetic management of children with anticipated difficult intubation should always include a full and frank discussion of risks with parents (and child if appropriate). The possibility of tracheostomy in case of failure to secure the airway should always be mentioned.

In adult anesthetic practice, an awake technique is often employed, but this cannot be done in children since cooperation is required to gain good bronchoscopic views. Antisialogogue premedication should be administered and child should be anesthetized maintaining spontaneous ventilation. Here due to a big disfiguring mass, achieving proper mask fit may be difficult. Oxygen should be given with either sevoflurane or halothane. Intubation should be performed under deep inhalational anesthesia. Alternately, anesthesia may be induced *using propofol taking care to preserve spontaneous breathing*. Use of a muscle relaxant during induction of anesthesia should be avoided as it may result in a situation of cannot-ventilate-cannot intubate (CVCI), necessitating establishment of surgical airway rapidly.

Merits and demerits of inhalational induction versus intravenous induction in a patient with difficult airway: Inhalational induction assures maintenance of spontaneous breathing which is crucial in difficult airway situations. Also patient regulates his/her own depth of anesthesia—in case the obstruction worsens with deepening plane, less inhalational agent will be delivered and patients depth of anesthesia will be reduced bringing back muscle tone and establishing patency of airway. However, inhalational induction is slow, may be irritant to airway, increase secretions, difficult to induce when proper mask fit cannot

be achieved, will lead to OT pollution, patient might become very agitated and difficult to manage in lighter plane and more importantly gives you very less time for laryngoscopy. As against this IV induction with propofol, is smooth, easily accepted by patient, one can easily control both the speed of induction and the depth of anesthesia and thus give more time for laryngoscopy. However, major problem is that there are chances of apnea, which can prove life-threatening.

For our case:

Plan A: Inhalational induction, laryngoscopy and intubation.

Plan B: *Fiber-optic bronchoscope-guided intubation under inhalational anesthesia*—The smallest size of FOB available has an outer diameter of 2.5 mm and thus can take a size 3.0–3.5 endotracheal tube loaded onto them which can be used even in infants. However, these scopes have no suction channel and secretions must be aspirated with a normal suction catheter. It is also a delicate piece of equipment and can be easily damaged. The nasal route is favored in adult practice and can be used successfully in children. Walker et al. have described the technique for difficult pediatric intubation using laryngeal mask airway (LMA). Inhalation induction with spontaneous ventilation is followed by the use of a standard LM to maintain depth of anesthesia and provide a conduit for the FOB. Guide the bronchoscope into the trachea through LMA. Pass a guidewire through the suction channel of the bronchoscope, remove the bronchoscope, and pass an airway exchange catheter over the guidewire. Correct positioning of the catheter is confirmed by capnography and visually via the bronchoscope. Finally, remove the LM and pass tracheal tube over the catheter into the trachea.

Alternatively, anesthesia can be induced using inhalational induction with sevoflurane. Once the child is anesthetized and breathing spontaneously, through one nostril a nasal trumpet (which is a nasal airway or a small ETT used as a nasal airway) can be connected to an anesthesia circuit and depth can be maintained using sevoflurane and spontaneous anesthesia. Through the other nostril a FOB-guided intubation can be done.

In the above mentioned case, insertion of LMA may be difficult due to big intraoral extension of mass. Either performing FOB-guided intubation or surgical tracheotomy in spontaneously breathing anesthetized patient; these are the two options that can be followed.

Plan C: Tracheostomy under inhalation anesthesia.

Case 4: A 70-year-old man, chronic bidi smoker (60 packs/year) presents with hoarseness and dysphagia of 6 months' duration. Since last 2 months he has difficulty in breathing and at present has mild inspiratory stridor. On flexible fiber-optic laryngoscopy, he has a large exophytic left pyriform fossa mass. He has been posted for direct laryngoscopy

to evaluate the extent of the lesion and biopsy to make a tissue diagnosis so that further plan of management can be decided. On airway examination, we find mouth opening 5 cm, Mallampati class I, and he has artificial dentures. Thyromental distance is three fingerbreadths and has normal range of neck movements. How will you manage airway?

Lesions arising in the hypopharynx are most frequently located in the pyriform fossa and can commonly grow to a size sufficient to cause airway obstruction. Though not very vascular, they are friable and tend to break in pieces with instrumentation. Large tumors also distort the anatomy in hypopharynx making identification of structures difficult, especially in the presence of blood. A narrowed airway then becomes completely occluded making mask ventilation also difficult.

Such patients who already have signs of stridor would not cooperate for awake fiber-optic laryngoscopy. Trying any airway manipulation in an uncooperative patient will further add to trauma, bleeding, and chances of dangerous airway obstruction.

Since this patient has partially obstructed airway, keep small-sized endotracheal tubes ready. Keep a tracheostomy standby and have low threshold to perform tracheostomy.

In this case choice of plans can be:

Plan A: Preoxygenate well, anesthetize with inhalational agents, perform gentle laryngoscopy and intubate if laryngoscopic view is good.

Plan B: Preoxygenate well, anesthetize with inhalational agents, perform gentle laryngoscopy and if you fail to have good laryngoscopy view, ask surgeons to perform direct laryngoscopy. As surgeons direct laryngoscope (**Fig. 4B**) is channeled and is longer in length visualization of glottis and intubation is much easier.



Fig. 4B: Direct laryngoscopy.

Plan C: Upfront tracheostomy and then proceed with procedure.

Alternatively these patients can be induced with IV propofol and follow above plans.

Remember that airway resistance is directly proportional to flow rates and inversely proportional to size of glottic opening. In the presence of anxiety patient tries to hyperventilate, thus increasing inspiratory flow rate and getting feeling of more breathlessness. With induction of anesthesia, flow rates fall and one might find that glottis is not that badly narrow.

All blind intubation techniques are contraindicated in this setting with distorted anatomy, friable tumor and potential for trauma.

Case 5: A 53-year-old female patient weighing 55 kg, presents with history of neck swelling gradually increasing over last 5 months with change in voice since 4 months and dysphagia to liquids and solids since 1 month. She also gives history of choking sensation intermittently. CT scan showed enlarged thyroid, with tracheal compression and mediastinal extension. Hopkins mirror examination showed left vocal cord fixed. Her thyroid function hematology and biochemistry investigations are normal. How will you manage her airway?

Large goiters or cancers of the thyroid can be a threat to the airway by external airway compression, deviation, or distortion. Such lesions can cause upper airway obstruction by local invasion of the trachea, softening of the trachea (tracheomalacia), mass effect causing distortion of the trachea, and through involvement of the recurrent laryngeal nerve. Involvement of this nerve may jeopardize an already compromised airway by causing additional narrowing of the glottic opening as in our case.

Routine preoperative indirect laryngoscopy to evaluate the vocal cords of all patients presenting for thyroid surgery is important for both medical and medicolegal reasons. Medically it is important to detect nerve palsy which may not be manifest by voice changes because of compensation by the nerve on the other side. This would emphasize the importance of special caution in terms of preservation of the contralateral nerve. Medicolegally, such evaluation demonstrates the presence or absence of nerve involvement preoperatively, a fact which is of importance in the evaluation of voice disturbance postoperatively.

All patients with an anterior mediastinal mass should have a chest radiograph and a CT scan prior to any surgical procedure and the anesthesiologist must look at the imaging to plan the airway management. The CT scan will show the site, the severity, and the extent of the airway compromise. *Most important in such situations is not to allow loss of muscle tone, as that may be what is keeping tumor off the airway.*

Most of the times visualization of glottis on direct laryngoscopy is not a problem as problem lies below

vocal cord level but correct placement of distal end of tube is difficult. Awake fiber-optic intubation is a safe and expeditious approach to the airway in a patient of this type. The major advantage of FOB-guided intubation is that one can place distal end of tube beyond the level of obstruction.

Patients with an anterior mediastinal mass who require GA need a step-by-step induction of anesthesia with continuous monitoring of gas exchange and hemodynamics. Maintaining spontaneous ventilation until the airway is definitively secured is a safest and popular strategy. Anesthetic induction can be inhalational with a volatile agent such as sevoflurane, or by IV titration of propofol, with or without ketamine. If muscle relaxants are required, assisted ventilation should first be gradually taken over manually to assure that positive-pressure ventilation is possible and only then can a short-acting muscle relaxant be administered. Development of airway or vascular compression requires that the patient be awakened as rapidly as possible and then other options for surgery can be explored. Perioperative life-threatening airway compression usually responds to one of two therapies: either repositioning of the patient (it should be determined before induction if there is one side or position that causes less symptomatic compression) or rigid bronchoscopy to open the obstruction for which an experienced bronchoscopist and rigid bronchoscopy equipment must always be immediately available in the operating room during these cases. For patients with life-threatening cardiovascular compression after induction that does not respond to lightening the anesthetic the only therapy is immediate sternotomy and surgical elevation of the mass off the great vessels.

There are important differences in the management of airway in mediastinal tumors in children versus adults. Anesthetic deaths have mainly been reported in children. The deaths may be the result of the more compressible cartilaginous structure of the airway in children or because of underestimation of the severity of the airway compression in children due to the difficulty in obtaining a clear history of positional symptoms. Even with proper management, children with tracheobronchial compression >50% cannot safely be given GA. Also, securing the distal airway with awake fiber-optic intubation and placement of an endotracheal tube distal to a tracheal obstruction, which is an option for some adults with masses compressing the mid-trachea, is not an option in most children.

Q. What is the role of cardiopulmonary (CP) bypass in cases of mediastinal tumors with distal airway compression?

Ans. Many authors and textbooks advocate CP bypass as a “standby” during induction of anesthesia for patients with large mediastinal tumor. The establishment of CP bypass

prior to induction of anesthesia can be safely performed in adult patients. But once airway or cardiovascular collapse occurs, it will require at least 5–10 minutes to cannulate and establish adequate circulation and oxygenation, even with a primed pump and a prepared team. In such a scenario, it is probable that a young patient can be resuscitated but will suffer neurological injury. Patients with severe symptoms due to large mass and airway or cardiovascular compression should not be given GA, even with maintenance of spontaneous ventilation. In these patients CP bypass or ECMO should be established prior to induction of anesthesia to maintain oxygenation or circulation.

Case 6: A 72-year-old man presented for coronary artery bypass grafting (CABG) for triple vessel disease. Airway evaluation showed mouth opening 4–5 cm, Mallampati II and prominent upper front teeth and incisors. Thyromental distance is three fingerbreadths. He has normal neck movements. Induction of anesthesia was achieved with thiopental, and vecuronium and patient could be adequately mask ventilated with oxygen, and isoflurane. On direct laryngoscopy only posterior arytenoids could be visualized but even after four attempts, could not be intubated. How will you tackle this situation?

This is a situation of unanticipated difficult intubation: Elective Situation—unanticipated, failed intubation following induction of GA is one of the most common airway emergencies anesthesiologists encounter in the operating room. What should one do when initial attempts at intubation are unsuccessful? The initial management following the first failed attempt is most important because the manner in which the initial problem is handled may greatly influence the subsequent outcome of this potentially life-threatening emergency.

Failed first attempt:

- Ventilate with 100% O₂
- Call for help
- Note time.

Maintain adequate anesthesia: Use volatile inhalational agents with oxygen as that will still help in using high FiO₂ and maintain adequate depth of anesthesia. Downside is “this might lead to OT pollution”. Alternatively, one can use repeated boluses/infusion of IV hypnotic agents:

- Check vital signs
- Reassess head and neck position
- Define the problem.

Reassess equipment selection: Change the technique and equipment with which you are familiar. There is no point in insisting on the same technique with which you have failed. One can use gum elastic bougie, different blades, LMA, ILMA, etc.

Get the most skilled person available.

The next attempt has to be the “best” attempt (you had already four unsuccessful attempts, actually one has to stop at two attempts and not get tempted to have one more go), i.e. optimal neck position, optimal laryngoscope blade, optimal external laryngeal manipulation, release cricoid pressure, use of stylet and gum elastic bougie and the most experienced airway expert. A videolaryngoscope may also be used. Until all these are ready it is best to keep oxygenating and ventilating the patient rather than multiple attempts at intubation.

Difficult Airway Society guidelines advocate only three attempts at laryngoscopy. This may seem a drastic step, allowing only three attempts at visualization; however, a little reflection will reveal that multiple unsuccessful attempts at intubation can change a controlled situation into an uncontrolled one. Airway structures including the tongue, pharynx, and the epiglottis are delicate structures, which are very prone to injury, bleeding and swelling, all of which impair visualization, making the laryngoscopic view more difficult and worse, impairing mask ventilation, leading to the life-threatening cannot intubate/cannot oxygenate (CICO) situation.

The All India Difficult Airway Association (AIDAA) 2016 guidelines recommend giving nasal oxygen at 15 L/min during attempts at laryngoscopy for apneic oxygenation (discussed later). Also subsequent laryngoscopies should be attempted only if the oxygen saturation is higher than 95%.

If the further attempt fails:

In an elective situation like this one should prefer to wake the patient and cancel surgery. Subsequently patient can undergo FOB-guided intubation, or retrograde intubation.

In an emergency situation one can use an LMA/ILMA, and proceed with surgery if safe (no aspiration risk, well-fitting LMA, surgery in supine position, normal airway pressures) or perform a surgical tracheostomy.

Case 7: Weight 80 kg, a 62-year-old male patient presents for cholecystectomy. On airway evaluation mouth opening is 6 cm. Mallampati I. Absent left lateral incisor and canine. Thyromental distance-three and half-finger breadths and has normal neck movements. No airway problems were anticipated preoperatively by either the surgeon or anesthesiologist. Patient was induced with fentanyl, thiopental, and succinylcholine. Oxygenation and ventilation was maintained by mask and two attempts were made to intubate the patient, neither successful. There was dislodgment of tooth and considerable bleeding after the first attempt. Patient could be ventilated with mask. First operator again tried twice but was unsuccessful. A second anesthesiologist attempted to intubate the patient but failed on further two attempts to visualize even the arytenoids. Mask ventilation after this could not be established and

there was significant stomach distension, there was considerable bleeding, the oropharynx suctioned but patient could not be ventilated and the saturation begins to fall: 70....60....50.....40%.

This is a situation of unanticipated difficult intubation: Emergency CICO situation.

Remember patients usually do not die from our failure to intubate; they die because we cannot stop trying till we land up in situation like this. In this case with bleeding at first attempt the operator should have maintained mask ventilation and reassess the situation. Change of technique or change of operator is dictum for failed first attempt. Bleeding and edema are progressive with each attempted intubation and a patent airway can be lost as happened here. In the life-threatening CICO situation, the DAS guidelines mention three steps for proceeding as mentioned in the **Flowchart 1**.

Step A: After the failed attempts at intubation.

Step B: Insertion of a SAD:

- If successful then wake up patient, continue case under SAD, attempt intubation through SAD with FOB or perform a tracheostomy under anesthesia.
- If unsuccessful proceed to step C.

Step C: One more attempt at rescue facemask ventilation using airway adjuncts:

- If successful continue oxygenation and ventilation till patient wakes up.
- If Unsuccessful proceed to step D.

Step D: Emergency front of neck access (FONA)

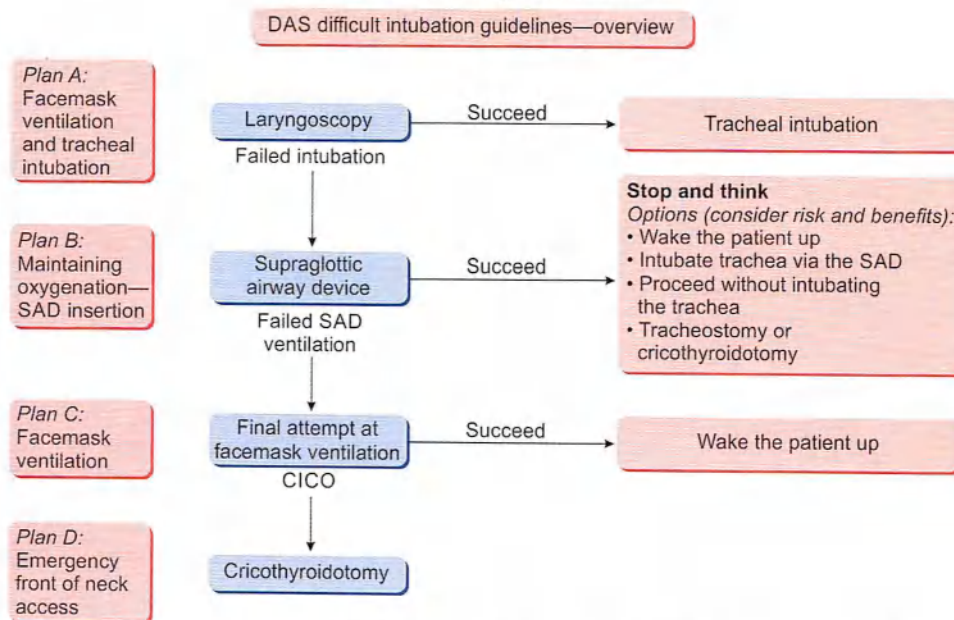
Three techniques have been described for FONA:

1. Surgical cricothyrotomy
2. Needle cricothyrotomy with high pressure jet ventilation devices
3. Cannula cricothyrotomy with wide bore (>4 mm) commercial cannula sets (Seldinger and non-Seldinger sets). Following the NAP4 audit the DAS 2015 guidelines recommend surgical cricothyrotomy as the technique of choice in terms of success rate.

Surgical Cricothyrotomy

- Commonly described as scalpel technique: “stab, twist, bougie, tube”.
- The laryngeal handshake is used to identify the thyroid cartilage, the cricothyroid membrane is identified moving the fingers more caudally from the thyroid notch.
- Make a transverse stab incision through cricothyroid membrane using a 10-No. scalpel blade.
- Rotate scalpel so that sharp edge points caudally.
- Pull the scalpel toward you to open up the incision.
- Slide coude tip of bougie down scalpel blade into trachea.
- Railroad 6 No. endotracheal tube into the trachea.

Flowchart 1: DAS guidelines 2015.



(CICO: cannot intubate/cannot oxygenate; DAS: Difficult Airway Society; SAD: supraglottic airway device).

Needle cricothyrotomy with transtracheal jet ventilation (TJV) in CVCI situation (narrow cannula <4 mm): A technique to establish TJV should be part of every anesthetist's clinical skill and the equipment necessary to do so should be included in every difficult airway cart. The technique should be considered early in all situations where nonsurgical techniques have failed to establish adequate ventilation. The TJV technique, using needle cricothyrotomy, is perhaps the fastest method to establish oxygenation in a patient of CICO.

In adults, this is performed by placing a large-caliber cannula 12- to 14-gauge, through the cricothyroid membrane into the trachea. The normal cannulae used for gaining IV access are not a good choice as they are susceptible to kinking and due to larger length offer higher resistance to flow. Commercially available needles like the Ravussin needles are kink resistant and have shorter length. These teflon cannulae are the preferred ones and are available in Indian market today. After confirming correct placement by aspirating air in a saline filled syringe, the needle is removed and the cannula fully inserted into the trachea in a caudal direction and firmly held.

The cannula is then connected to a pressure regulated jet injector such as the Manujet and Sanders jet injector. It is advisable to start with the lowest pressure on these devices and gradually increase the pressure till you get a visible chest rise and the oxygenation of the patient improves (maximum pressure 60 psi). The upper airway should be kept patent to allow for expiration to occur. Adequate time for expiration should be given while using jet ventilation

(I:E ratio 1:4). Remember that in patients with completely obstructed upper airway expiration occurs through very narrow lumen of inserted cannula and one needs to give adequate time for expiration to prevent barotrauma. Needle cricothyrotomy is only a rescue airway and a definite airway has to be established in form of tracheostomy as early as possible.

Wide Bore Cannula Cricothyrotomy (>4 mm)

Today, several ready-to-use commercial kits are available in the market for performing cricothyrotomy. These are the PCK, Pertrac, Quicktraq (non-Seldinger technique). The Seldinger technique (Cook Melker set) is associated with maximum success and least morbidity. This kit includes 4.0 mm cuffed ID tube, a needle, syringe, flexible wire, and No. 10 scalpel blade.

The advantages with a wide bore cricothyrotomy is that we can ventilate effectively due to cuffed tube with anesthesia breathing circuits, and the chances of barotraumas and aspiration are less as compared to a needle cricothyrotomy. Capnography can also be used with a cannula cricothyrotomy but not with a needle cricothyrotomy device.

Role of Combitube in Cannot-Ventilate-Cannot Intubate Situation

Combitube is a disposable double lumen tube that combines the features of a conventional tracheal tube and that of an esophageal obturator airway (details are given in instrument section). Parmet et al. have reported of rescuing 16 of 17 CVCI patients with use of Combitube. The only

patient who could not be rescued by them was a patient with intratracheal blood clots. Davis et al. have shown 97–99% success rate of using Combitube in prehospital airway rescue when patients could not be intubated. It is seldom used outside of the prehospital, emergency setting, as it does not allow for long-term airway control. With the availability of better airway gadgets especially better SGAs role of Combitube even in prehospital setting has declined.

Case 8: A 35-year-old obese male patient comes for an elective gallbladder surgery. The saturation on room air in the supine position is 92%. Discuss how you can improve oxygenation and prevent desaturation during laryngoscopy and increase the apnea time.

Obese patients have a tendency to desaturate quickly as the closing capacity is higher than the functional residual capacity (FRC). Apnea time for laryngoscopy can be prolonged in these patients by adequate preoxygenation, using continuous positive airway pressure (CPAP) of 5–10 cm of water for preoxygenation, propped up and ramp position and giving nasal oxygen during laryngoscopy @15 L/min flow rate with nasal prongs.

This technique of nasal oxygen (**Fig. 5**) was described by Richard Levitan as NO DESAT (nasal oxygen during efforts securing a tube), which uses a simple nasal cannula with standard low-flow cold dry oxygen at a rate of 5–15 L/min. NO DESAT allows apneic oxygenation to continue while attempts at tracheal intubation are performed, and it increases apnea times by 2–5 minutes.



Fig. 5: Technique of nasal oxygen.

Apneic oxygenation is a physiologic phenomenon in which alveoli will continue to take up oxygen without any diaphragmatic movements or lung movement, through a patent airway. The difference between the alveolar rates of oxygen removal (200–250 mL/min) and CO₂ excretion into the alveoli (~20 mL/min) generates negative pressure up to 20 cm H₂O. This generates a mass flow of gas from the

pharynx to the alveoli, which drives oxygen into the lungs. The application of low-flow oxygen (15 L/min) through a nasal cannula increases pharyngeal FiO₂, which acts as a pharyngeal oxygen reservoir that leads to a greater amount of oxygen delivery during the mass flow of gas from the pharynx to the alveoli. This maintains oxygenation for longer and increases apnea time, but will have little effect on CO₂ clearance, resulting in hypercapnia and acidosis.

Q. What are the prerequisites of apneic oxygenation?

Ans.

- High oxygen concentration:** Apneic oxygenation can be effectively performed following preoxygenation to ensure high oxygen concentrations throughout the respiratory tree. This is required to ensure that the net volume of gas being taken up from the alveoli is replenished with gas from the upper airway that contains sufficient oxygen to maintain adequate oxygenation of pulmonary capillary blood and allow the process to continue. Due to this reason, apneic oxygenation is not effective in restoring the oxygen saturations in patients who have already desaturated that happens during airway management.
- Oxygen source:** Insufflation of high-flow oxygen into the upper airway via the nasal or buccal routes prevents entrainment of room air in order that high concentration oxygen is available to move down the trachea to the alveoli and replace that being consumed.
- Patent upper airway:** Oxygen delivered to the upper airway can only move along the pressure gradient to lungs only if the upper airway is patent. This is one of the most important prerequisites for a successful apneic oxygenation.

Q. What are the limitations of apneic oxygenation?

Ans. An important point to remember is that apneic oxygenation is an important adjunctive tool to extend the safe apnea time but that it cannot be used as an alternative to the definitive strategy of restoring alveolar oxygen delivery. Though apneic oxygenation prolongs the safe apnea time significantly, this is not guaranteed and some patients such as obese and patients with preexisting shunt physiology may still rapidly desaturate despite these techniques. Apart from airway obstruction, alveolar collapse affects the efficacy of apneic oxygenation. Apneic oxygenation has very little effect on CO₂ clearance and leads to hypercapnia and acidosis.

Q. What are the different techniques of apneic oxygenation?

Ans.

- NO DESAT (nasal oxygen during efforts securing a tube):** The application of low-flow oxygen (15 L/min) through a nasal cannula increases pharyngeal FiO₂, which acts as a pharyngeal oxygen reservoir that leads to a greater

amount of oxygen delivery during the mass flow of gas from the pharynx to the alveoli. Using nasal cannulae at 15 L/min, apneic oxygenation has been demonstrated to extend the duration of apnea without desaturation to beyond 10 minutes in some cases.

- b. **THRIVE (transnasal humidified rapid-insufflation ventilatory exchange):** THRIVE is a technique that uses rapidly insufflated, heated, humidified oxygen administered via HFNC to achieve apneic oxygenation and ventilation. HFNC provides oxygen at flows much higher than the patient's minimum ventilation and allows titration of delivered O_2 up to 100%. These high flows produce a distending alveolar pressure, or positive-end expiratory pressure (PEEP), up to 7 cm H_2O , which prevents airway collapse and atelectasis. By providing humidified warm air, HFNC limits mucosal injury, ciliary dysfunction, infection, and patient discomfort. THRIVE may be ineffective or dangerous in patients with severe obstructive upper airway pathology, congenital airway anomalies, elevated ICP, patients at high risk of aspiration, hemodynamically unstable patient. The greatest advantage of THRIVE appears to be its potential to provide ventilation. Some authors have suggested that there is likely significant ventilation and gas exchange as a result of gaseous mixing and flushing of dead space secondary to the high insufflation flows with HFNC. Each company offers smaller cannula sizes for premature neonates as well as children of various ages to adult cannulas. The device consists of nasal cannulae with standard sized or wide-bore prongs connected to an oxygen flow meter with an air-oxygen gas blender and gas analyzer. They offer maximum gas flow rates of between 40

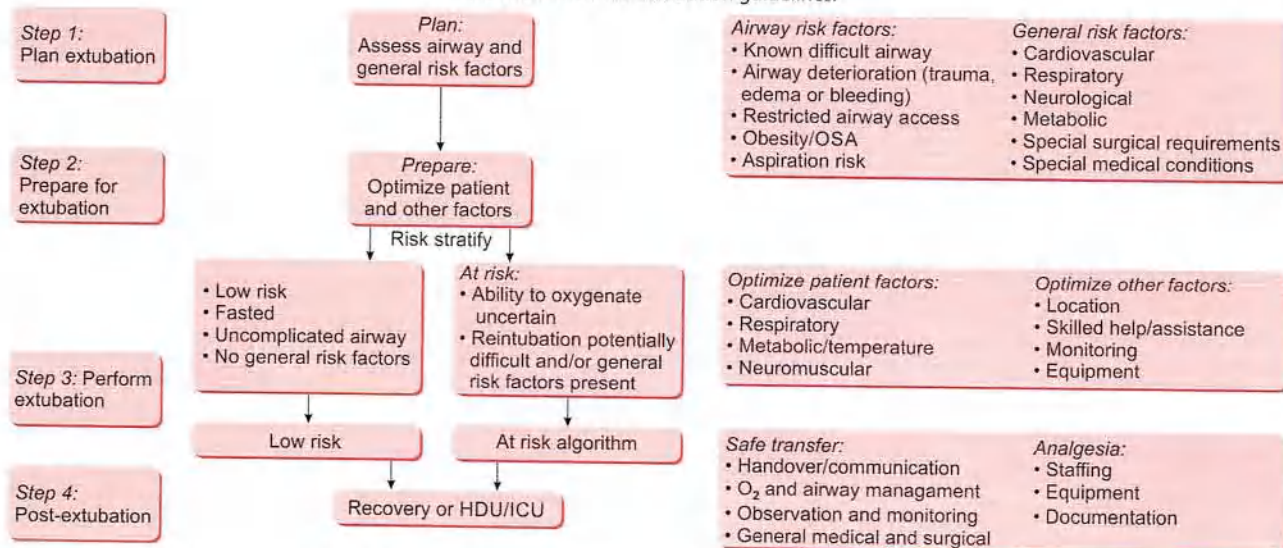
and 60 L/min, depending on the device. A heating system and humidifier allows delivery of gases at temperatures of between 33 and 43°C and 95–100% humidity.

Case 9: A 73-year-old man underwent complex facial surgery with radial forearm free-flap reconstruction, and shifted to recovery room with nasal ETT in situ to be extubated coming morning. Four hours after shifting he self-extubated and has partial airway obstruction. How will you manage?

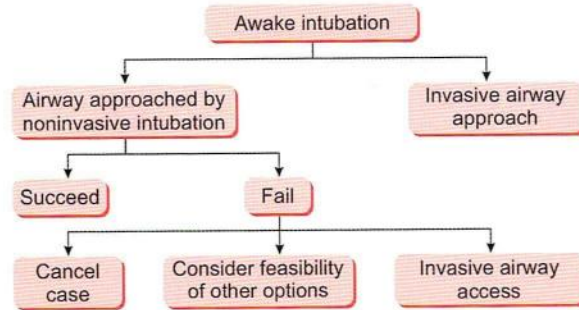
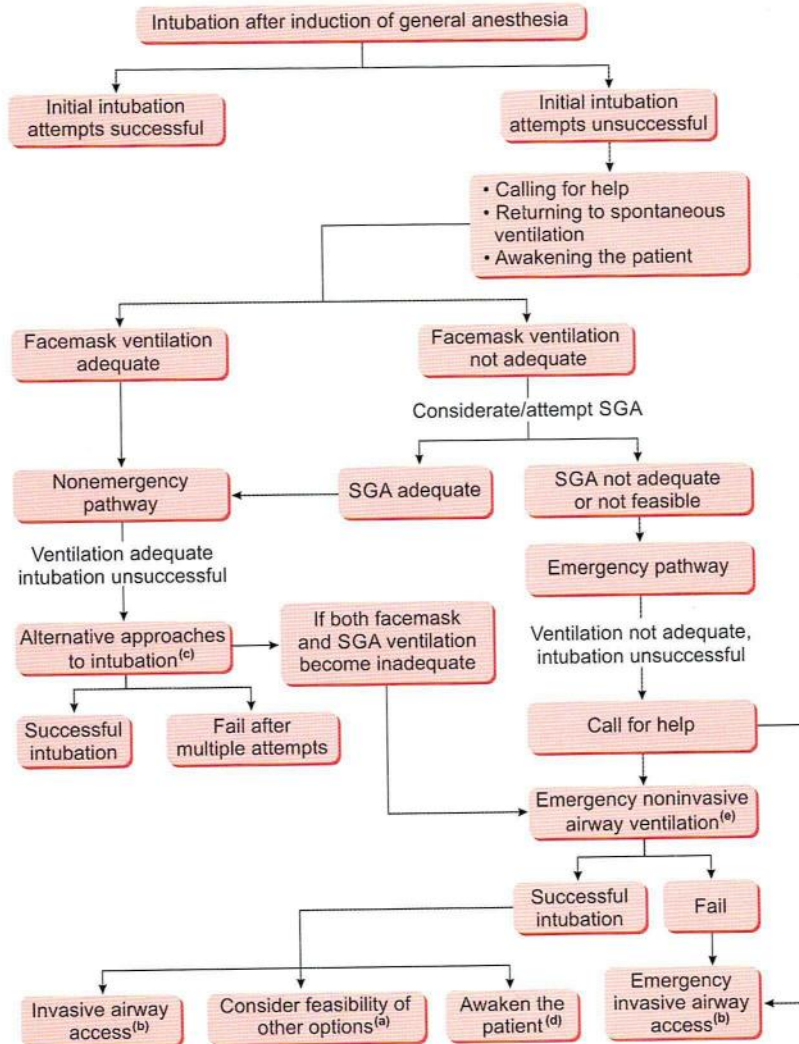
The moment you realize that your patient has self-extubated, call surgeons for your help as situation can turn tricky. Oxygenate the patient. Here patient has fresh surgery with intraoral reconstruction which has changed the anatomy. There will be intraoral edema, secretions and some amount of blood. Patient might be having airway obstruction due to secretions which he is unable to clear. Perform a thorough intraoral suction. Try and oxygenate the patient with mask. Any attempts of direct laryngoscopy in uncontrolled environment can be disastrous. Possibility of a retained gauze piece or throat pack should be considered and rechecked with surgeons. Doing emergency tracheostomy is not an easy option here as patient will be uncooperative, achieving proper position will be difficult, and using sedation to get patients cooperation will be faced with danger of losing the existing airway. It would be preferable to shift this patient back to OT, upper airway can be nebulized with 4% lignocaine and try and perform gentle FOB and intubate (**Flowcharts 2 to 4 and Box 1**).

The AIDAA 2016 extubation guidelines recommend the use of pharmacological agents during extubation to blunt the hemodynamic stress response to extubation. Alternatively exchange with a supraglottic device (Archie's

Flowchart 2: DAS extubation guidelines.



(DAS: Difficult Airway Society; HDU/ICU: high dependency unit/intensive care unit; OSA: obstructive sleep apnea).

Flowchart 3: Awake intubation.**Flowchart 4: Intubation after induction of general anesthesia.**

(SGA: supraglottic airway).

a. Other options include (but are not limited to): Surgery utilizing facemask or supraglottic airway (SGA) anesthesia (e.g., LMA, ILMA, laryngeal tube), local anesthesia infiltration or regional nerve blockade. Pursuit of these options usually implies that mask ventilation will not be problematic. Therefore, these options may be of limited value if this step in the algorithm has been reached via the emergency pathway.

b. Invasive airway access includes surgical or percutaneous airway, jet ventilation, and retrograde intubation.

c. Alternative difficult intubation approaches include (but are not limited to): Video-assisted laryngoscopy, alternative laryngoscope blades, SGA (e.g., LMA or ILMA) as an intubation conduit (with or without fiber-optic guidance), fiber-optic intubation, intubating stylet or tube changer, light wand, and blind oral or nasal intubation.

d. Consider rep preparation of the patient for awake intubation or canceling surgery.

e. Emergency noninvasive airway ventilation consists of a SGA.

manoeuvre), airway exchange catheter and ventilator leak test may be done depending on the patient's airway.

BOX 1: ASA 2013 algorithm.

- Assess the likelihood and clinical impact of basic management problems:
 - Difficulty with patient cooperation or consent
 - Difficult mask ventilation
 - Difficult supraglottic airway placement
 - Difficult laryngoscopy
 - Difficult intubation
 - Difficult surgical airway access
- Actively pursue opportunities to deliver supplemental oxygen throughout the process of difficult airway management
- Consider the relative merits and feasibility of basic management choices:
 - Awake intubation versus intubation after induction of GA
 - Noninvasive technique versus invasive techniques for the initial approach to intubation
 - Video-assisted laryngoscopy as an initial approach to intubation
 - Preservation versus ablation of spontaneous ventilation
- Develop primary and alternative strategies.

(ASA: American Society of Anesthesiologists).

Q. How is tracheal intubation in the ICU different from tracheal intubation in the operating room?

Ans. Intensive care unit intubation protocol: In ICU, critically ill patients with respiratory failure and shock are physiologically unstable and are described best as a physiologically difficult airway. Thus, their suboptimal response to preoxygenation is responsible for a high incidence of life-threatening complications such as severe hypoxemia and cardiovascular collapse during tracheal intubation in ICU. The AIDAA recommends a stepwise plan for airway management in critically ill patients. Following considerations have been highlighted by AIDAA during tracheal intubation in ICU:

- AIDAA recommends the presence of two operators during ICU intubations, with one being experienced in airway management.
- It is recommended to call for additional help early when the first difficulty in airway management is encountered. Also, call for additional person, who has not been part of the process until then when the final attempt at rescue mask ventilation fails and emergency cricothyroidotomy is planned.
- Proper preoxygenation is needed to prolong the safe apnea time during intubation and increase the chances of success at the first attempt of intubation. After preoxygenation with noninvasive positive pressure ventilation, the addition of apneic oxygenation during intubation attempts significantly reduces the severity of desaturation.
- Adequate fluid resuscitation, prompt treatment of postintubation hypotension and proper selection of drugs for intubation are important to prevent postintubation hypotension.

- It is important to train ICU staff about various advanced airway equipment's and make appropriate devices of airway management available in the ICU.
- Rapid sequence induction with either suxamethonium or rocuronium and cricoid pressure is recommended in ICU patients. However, if the cricoid pressure makes the laryngoscopic view difficult, it should be released. A second-generation SAD should be inserted if intubation fails during a rapid sequence induction.
- It is strongly recommended to use capnography for confirmation of tracheal intubation in all critically-ill patients, irrespective of location.
- Airway management training should be an integral part of the critical care training. Use of an algorithm and an intubation bundle may further improve performance.
- In an ICU patient unlike an operating room patient for elective surgery waking up the patient in case of failed intubation is not an option. The options available are emergency cricothyrotomy followed by a tracheostomy as early as possible.

Following the AIDAA guidelines, DAS has also laid down guidelines for tracheal intubation in the intensive care unit. The salient features are similar to those described above.

SUGGESTED READING

1. Ahmad I, El-Boghdady K, Bhargava R, Hodzovic I, McNarry AE, Mir F, et al. Difficult Airway Society guidelines for awake tracheal intubation (ATI) in adults. *Anaesthesia*. 2019. doi: 10.1111/anae.14904. [Epub ahead of print]
2. All India Difficult Airway Association. 2016 Airway Guidelines. *Indian J Anaesth*. 2016;60:883-930.
3. Apfelbaum JL, Hagberg CA, Caplan RA, Blitt CD, Connis RT, Nickinovich DG, et al. American Society of Anesthesiologists Task Force on Management of the Difficult Airway. Practice guidelines for management of the difficult airway: an updated report by the American Society of Anesthesiologists Task Force on Management of the Difficult Airway. *Anesthesiology*. 2013;118:251-70.
4. El-Orbany M, Woehlck HJ. Difficult mask ventilation. *Anesth Analg*. 2009;109:1870-80.
5. Frerk C, Mitchell VS, McNarry AE, Mendonca C, Bhargava R, Patel A, et al. Difficult Airway Society intubation guidelines working group. Difficult Airway Society 2015 guidelines for management of unanticipated difficult intubation in adults. *Br J Anaesth*. 2015;115:827-48.
6. Kheterpal S, Han R, Tremper KK, Shanks A, Tait AR, O'Reilly M, et al. Incidence and Prediction of Difficult and Impossible Mask Ventilation. *Anesthesiology*. 2006;105:885-91.
7. Patel A, Nouraei SA. Transnasal Humidified Rapid-Insufflation Ventilatory Exchange (THRIVE): a physiological method of increasing apnoea time in patients with difficult airways. *Anaesthesia*. 2015;70:323-9.
8. Popat M, Mitchell V, Dravid R, Patel A, Swampillai C, Higgs A. Difficult Airway Society Extubation Guidelines Group. Difficult Airway Society Guidelines for the management of tracheal extubation. *Anaesthesia*. 2012;67:318-40.
9. Yentis SM. Predicting difficult intubation—worthwhile exercise or pointless ritual? *Anaesthesia*. 2002;57:105-9.

Nayana Amin, Vijaya Prakash Patil, Bhakti Trivedi

A 30-year-old male patient is brought to the casualty after being rescued from a house fire. He has burns over his face, hands, and trunk.

Q. How will you assess the severity of burns?

Ans. The severity of burns can be assessed in several ways. There are three commonly used methods for measuring a burn:

1. Wallace rule of nines is used to assess the percentage of body surface area (BSA) burnt. The total body surface area (TBSA) is divided into multiples of nine and is used as a rule of thumb to quickly calculate the TBSA burnt. Though it tends to overestimate the burned area, it is a good method for measuring medium to large burns (**Fig. 1**).

Head and neck	9%
Upper extremities	9% each
Chest (anterior and posterior)	9% each
Abdomen	9%
Lower back	9%
Lower extremities	18% each
Perineum	1%

2. **Palmar surface:** The palmar surface of the patient's hand, with fingers very slightly spread, equates to approximately 1% of the patient's TBSA. This is a quick and reliable method of assessing patchy areas of burns up to 15% of TBSA. It is not very accurate in estimating moderate burns. If the extent of burns is >85%, then burns can be estimated by simply measuring the unburned areas.

3. Lund and Browder chart (described later).

Burns can also be classified according to the depth of the injury. Assessment of the depth of the burn is important for planning wound care and predicting functional and cosmetic results.

Superficial: Only epidermis is damaged. Heals spontaneously within 7 days with no scarring. There is erythema, no blisters, it blanches on pressure, and is painful.

Superficial partial thickness (superficial dermal): Epidermis completely destroyed, together with approximately the upper one-third of the dermis, but the adnexal skin structures (hair follicles, sebaceous glands, and sweat glands) survive. However, the epithelium regenerates rapidly and the wound heals within 10–14 days without any scarring. It has a red and mottled appearance with swelling and blanches on pressure. There is blister formation and is painful.

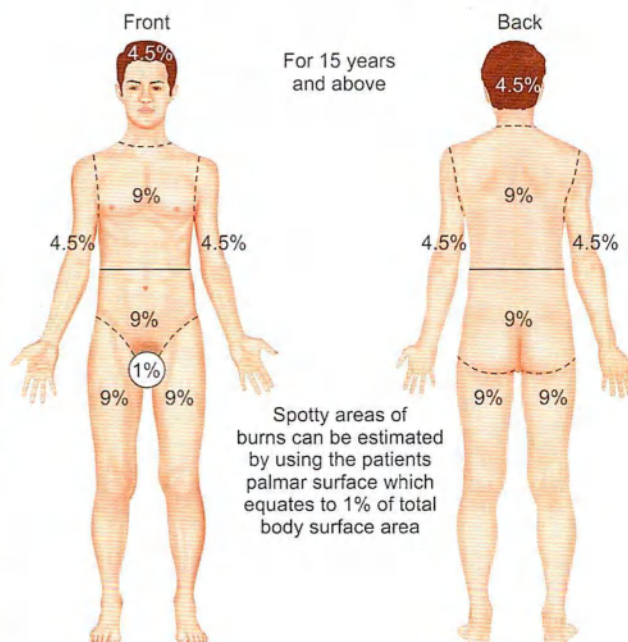


Fig. 1: Calculation of body surface area in adults.

Deep partial thickness (deep dermal): Epidermis and substantial part of dermis with adnexal structures are

destroyed. Healing takes several weeks or months as epithelialization is slow. There is scarring. It is pale in appearance with no blanching. There is no pain only pressure sensation is felt. These burns require excision and grafting for rapid return of function.

Full thickness: All skin elements destroyed. Damage may extend to involve deep structures such as muscle, bone, and major neurovascular bundles. It appears dark and leathery. The surface is dry and painless and does not blanch on pressure. It requires excision and grafting. Heals very slowly by wound contracture with minimal epithelialization from edges.

Q. Is the “rule of nines” also used for assessing the severity of burns in children?

Ans. Children have relatively larger heads and smaller limbs as compared to adults. Due to the different body proportions, the “rule of nines” is inaccurate in children. Age-adjusted nomograms are a better method because they reflect the gradual change in the relative BSAs with age. Currently, the Lund and Browder chart is the most accurate method for assessing the burn area in children (**Fig. 2 and Table 1**).

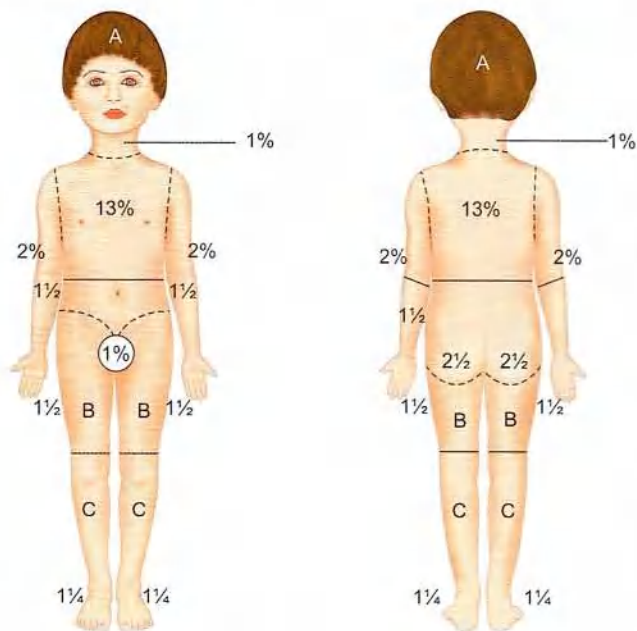


Fig. 2: Calculation of body surface area in children.

TABLE 1: Calculation of percentage of BSA burn in children (age in years).

Area	0	1–4	5–9	10–14	15
A: Half of head	9½	8½	6½	5½	4½
B: Half of one thigh	2¾	3¾	4	4¼	4¼
C: Half of one lower leg	2½	2½	2½	3	3¼

(BSA: body surface area).

Q. Briefly describe the three major layers that make up healthy skin.

Ans.

1. **Epidermis:** This layer is composed of keratinized, stratified, and squamous epithelium. It provides a thick, water-proof, and protective covering over the underlying layers of skin.
2. **Dermis:** This layer is composed primarily of dense, irregular, fibrous connective tissue that is rich in collagen (for strength) and elastin (for elasticity). The dermis contains blood vessels, nerve endings, and epidermally derived cutaneous organs such as sweat glands, sebaceous glands, and hair follicles.
3. **Hypodermis (subcutaneous layer):** This layer is composed primarily of loose, areolar connective tissue and adipose tissue. This fat layer provides both mechanical cushioning as well as a thermal insulation for the underlying organs.

Q. What are the functions of skin?

Ans. Skin is the largest organ in the body. In adults, the surface area ranges from 1.5 to 2 m². It has various important functions:

- It protects the body against invasion from micro-organisms
- Temperature regulation
- Fluid and electrolyte homeostasis
- Touch, pain, and temperature sensations
- Vitamin D metabolism
- Ultraviolet (UV) protection.

Q. How would you determine the prognosis in a patient with burns?

Ans. Survival of a patient after burn injuries depends on the patient's age and percentage of BSA burnt. As a rough guide, % mortality = BSA of burn + Age (Baux score). Younger patients are more likely to survive. Other factors which influence the outcome are associated inhalational injury (doubles the mortality rate), depth of the burn, other injuries sustained during burns, and associated medical conditions.

Q. How would you define major burns?

Ans. Definitions of major burns are based on BSA burned and the area of the body burned:

- Third-degree (full thickness) burn injuries involving >10% of TBSA
- Second-degree (partial thickness) burn injuries involving >20% TBSA at extremes of age and >25% TBSA in adults
- Burns involving face, hands, feet, genitalia, perineum, and major joints
- Inhalational injuries
- Chemical burn injuries

- Electrical burn injuries
- Burn injuries in patients with coexisting medical disease
- Burns associated with trauma.

Q. What is the role of an anesthetist during the management of a burns patient?

Ans.

- Anesthetist may be involved in the initial resuscitation of a burns patient. Especially if there is hypoxemia and airway compromise which is life-threatening.
- Intensive care management would be needed if the patient develops sepsis and multiorgan failure.
- A burns patient would need general anesthesia for various surgical procedures:
 - Early excision of damaged tissues
 - Excision of granulation tissue and subsequent skin grafting
 - Change of dressings
 - Reconstructive plastic surgical procedures to relieve contractures, correct deformities, and restore limb function.

Q. What are the pathophysiologic changes seen in burn injuries?

Ans. Following burns, there is local and systemic inflammatory reaction, which results in the release of various inflammatory mediators (oxygen radicals, histamine, prostaglandin, bradykinin, nitric oxide, serotonin, and substance P and complement). In minor burns, the inflammatory process is limited to the wound itself. However, in major burns, local injury triggers the release of circulating mediators which results in a systemic response. Interleukins 1 and 6 and tumor necrosis factor (TNF) are the primary mediators of systemic inflammation after burns. Various studies have shown that endotoxin concentrations correlate with burn size and predict the development of multiple organ failure and death. After burn injury, there is increased nitric oxide levels which may contribute to hemodynamic and immunologic alterations.

Hemodynamic response: There is increased capillary permeability due to systemic inflammatory response which results in loss of large amounts of protein-rich fluids from the intravascular compartment to interstitial compartment. As a result of this, there is hypoalbuminemia which is further exacerbated by decreased hepatic albumin production in favor of acute phase protein synthesis. The loss of fluid into the interstitial space results in decreased circulating blood volume. Cell membrane adenosine triphosphatase activity is reduced which results in accumulation of intracellular sodium and water, further depleting the intravascular compartment. Fluid loss also continues by evaporation and exudation from the wound

site further causing intravascular depletion. During the first 24 hours of burns, there is release of catecholamines, vasopressin, and angiotensin which causes peripheral and splanchnic vasoconstriction and decreased cardiac contractility. All this can result in hypoperfusion of the end organs. This phase is known as burn shock. It is due to a combination of hypovolemic, cardiogenic, and distributive element. Excessive resuscitation at this stage leads to generalized edema, which further compromises tissue oxygenation. Reperfusion of the ischemic zone releases oxygen-free radicals further causing local cell membrane damage and immune response. After the patients are adequately fluid-resuscitated, there will be a phase of increased cardiac output and a marked reduction in systemic vascular resistance.

Respiratory responses: Release of inflammatory mediators can cause bronchoconstriction in the absence of inhalation injury. In patients with inhalational injury, the release of inflammatory mediators can lead to the development of the acute respiratory distress syndrome (ARDS).

Metabolic responses: After major burns, there is a hypermetabolic response which lasts for up to 1 year after the burns injury. There is decreased protein synthesis and increased muscle breakdown. Increased circulating levels of catabolic hormones lead to increased gluconeogenesis resistant to insulin infusion. Increased peripheral lipolysis leads to rapid depletion of glycogen stores. All these changes lead to impaired wound healing exposing patients to increased infection risk. Rehabilitation is often difficult in these patients.

Thermoregulation: Due to the hypermetabolic responses, the metabolic thermostat is reset. Burns patients increase their skin and core temperatures somewhat above normal regardless of the environmental temperatures. Following thermal injury, thermoregulatory functions of the skin such as vasoactivity, sweating, piloerection, and insulation are either abolished or diminished. Daily evaporative water loss is 2,500 mL/m² in adults and 4,000 mL/m² of burn surface in children.

Immune system responses: Following severe burns, there is loss of protective skin barrier and suppression of the immune system. Also, these patients need invasive lines and urinary catheter for monitoring. As a result of all this, burns patients have a significantly increased risk of repeated episodes of infection.

Hematologic system: Burns patients are usually anemic due to the loss of erythrocytes from the burn wound, bleeding during surgeries, hemolysis of erythrocytes due to thermal injury, suppression of erythrocyte production,

and decreased erythrocyte survival after burns. Also, the hematocrit is increased for at least 48 hours after burns due to hemoconcentration and may not be an appropriate guide for need for transfusion. Platelet counts and coagulation factors are decreased due to dilution and consumption. There is activation of thrombotic and fibrinolytic mechanisms.

Renal and electrolytes: Decrease in renal blood flow secondary to hypovolemia and decreased cardiac output and systemic vasoconstriction due to circulating catecholamines can lead to renal impairment. Other mechanisms for renal failure include the nephrotoxic effects of drugs, myoglobin, and sepsis. Renin-angiotensin-aldosterone system is activated due to decreased renal blood flow resulting in the release of antidiuretic hormone. The net effect on the renal function is retention of sodium and water and exaggerated losses of potassium, calcium, and magnesium. Hyperkalemia may occur due to tissue necrosis and hemolysis.

Gastrointestinal tract: Ileus is common in burns patients; hence, early decompression of the stomach through a nasogastric tube is indicated. Curling's ulcers are acute ulcerations of the stomach or duodenum seen in patients with sepsis or extensive burn injuries. The exact etiology is unknown. Endotoxemia can result from the disruption of the gastric mucosal barrier. Gastric mucosal ulcerations can be minimized by proper hemodynamic resuscitation, early enteral feeding, and the use of antacids and H_2 receptor blockers.

Q. You are called to the casualty to see a patient with burns. What would make you suspect the presence of inhalational injury?

Ans. The diagnosis of inhalational injury depends on a high degree of suspicion, clinical examination, and laboratory testing. Inhalational injury should be suspected if there is a history of explosion, impaired mentation and/or confinement in a burning environment, inhalation of toxic fumes, and associated alcohol intoxication.

On examination, there would be darkened or reddened oral and/or nasal mucosa, burns to the face, lips or nares, singed eyebrows, or nasal hairs. The presence of carbon or soot on teeth, tongue or throat, hoarse voice, or productive cough would indicate inhalational injury. Tracheal tug, inspiratory stridor, or inability to clear secretions may indicate impending airway occlusion. There may be swelling of the tissues around the airway due to circumferential burns to the neck. Therefore, early intubation is indicated in this situation. Arterial blood gases (showing high lactate levels in hemodynamically stable patient) and carbon monoxide (CO) levels (measured using CO-oximetry) may be useful in determining the degree of insult.

Q. What is the pathophysiology of inhalational injury?

Ans. Inhalation injury occurs as a result of direct thermal injury to the mucosa of the respiratory tract or from chemical tracheobronchitis due to the inhalation of incomplete products of combustion. The pathophysiological process leading to inhalational injury can be remembered using the mnemonic HOTT: Heat, Oxygen deficiency, Toxins—local, and Toxins—systemic.

Airway injury above the larynx: Direct thermal injury primarily occurs in the upper respiratory tract only. This is because there is reflex closure of the glottis and also because heat dissipation through the bronchial tree is very efficient. Usually, there is upper airway edema, which may progress to complete airway obstruction. The temperature required to produce such injury will depend on the heat capacity characteristics of the gas or vapor. Dry gases have less injurious potential than a similar exposure to saturated vapors. Upper airway edema usually peaks at 24 hours following injury and resolves within the first week. Stridor and respiratory distress indicate inhalation injury. If airway obstruction is suspected, intubation should be performed immediately as the pharyngeal mucosa swells rapidly, particularly after fluid resuscitation. Even patients with a mild upper airway injury are at risk for development of progressive airway obstruction as tissue edema develops.

Airway injury below the larynx: The lower airway is rarely burned by exposure to heat unless substances with a very high specific heat capacity are inhaled, such as superheated steam.

Chemical tracheobronchitis: The inhalation of incomplete products of combustion leads to a pulmonary inflammatory response similar to that seen after gastric acid aspiration. Water-soluble gases such as ammonia, nitrogen dioxide, sulfur dioxide, and chlorine combine with water in the respiratory tract, producing strong acids and alkalis. For example, sulfur dioxide forms sulfuric acid. Lipid-soluble gases such as aldehydes and hydrogen chloride are carried into the lower airways on carbon particles. Aldehydes such as acrolein, which are produced by the combustion of cotton, wood, and various synthetic fibers, impair ciliary function and damage mucosal surfaces. These chemical products act as direct irritants causing bronchospasm, edema, and mucous membrane ulceration. Necrotic cell debris, particulate matter, and proteinaceous fluid accumulate in the bronchial tree, leading to cast formation and atelectasis. The production of surfactant is impaired and a ventilation/perfusion mismatch develops as the alveolar injury evolves. The smaller the size of the smoke particle, the deeper is the penetration into the lung.

Systemic intoxication injuries: Toxins and chemicals such as CO, hydrogen cyanide, ammonia, hydrofluoric acid, and phosgene that reach the alveoli can be absorbed into the systemic circulation. Severe systemic intoxication often results in metabolic acidosis. CO interferes with tissue oxygenation by binding avidly to hemoglobin, preventing oxygen from binding to hemoglobin. It also binds to cytochrome oxidase and inhibits mitochondrial function at a cellular level. Hypoxic encephalopathy secondary to CO poisoning is thought to result from a reperfusion injury in which the products of lipid peroxidation and free radical formation contribute to morbidity and mortality. Combustion of plastics, polyurethane, wool, silk, nylon, nitriles, rubber, and paper products can all lead to the production of cyanide gas. Cyanide interferes with cellular metabolism by binding to the ferric ion in cytochrome A₃. Due to decreased oxygen utilization, there is anaerobic metabolism with development of lactic acidosis.

Oxygen deficiency: During inhalation injury, hypoxia is multifactorial and may be immediate or delayed:

- Utilization of oxygen during combustion can cause significant decrease in ambient oxygen concentrations.
- Loss of consciousness or airway edema can lead to airway obstruction.
- Inhalation of CO or hydrogen cyanide can result in cytotoxic hypoxia.

Lung injury develops in 24 and 48 hours resulting in delayed hypoxemia. This is significant as it usually coincides with the period of maximal tissue edema. The combination of low capillary oxygen tension and reduced tissue oxygen diffusion will compound tissue hypoxia, and subsequent "reperfusion" may result in worsening of the injury.

Q. What are the other components of smoke and their effects?

Ans.

- **Oxygen free radicals:** They cause peroxidation of the outer lipid layer of the cell leading to cell damage and cell death.
- **Ammonia:** It is an airway irritant causing cough, increased secretions, and bronchoconstriction. It forms ammonium hydroxide, a potent alkali, leading to tissue necrosis, especially of the lower airways.
- **Sulfur dioxide:** It is also an irritant to the airway and the eyes. It is oxidized to sulfurous acid and sulfuric acid. It is associated with lower airways injury and lung edema.
- **Nitrogen dioxide:** It is a gas with limited solubility in water and can be carried by particles. The injury is mainly to lower airways and is delayed in onset up to 72

hours. Lipid solubility leads to damage to all membranes and cell death.

- **Chlorine:** It is an irritant compound causing bronchospasm. When dissolved in water on the mucosal surface, it forms hydrochloride and hydrochloric acid. It is a potent oxidizing agent causing cell necrosis.

Q. How would you diagnose and treat inhalational injuries?

Ans. High-degree of suspicion based on the history, a good clinical examination, and laboratory testing will help in the diagnosis of inhalational injury (already discussed above).

The presence of inhalational injury is currently diagnosed by bronchoscopy or xenon lung scan. Fiberoptic bronchoscopy (FOB) is performed for diagnosis and treatment. Mild injury gives rise to erythema and edema of the mucosa. In severe inhalational injury, FOB may show gray-colored mucosa, erosions, ulcerations, and/or desquamation. However, there is no method available for assessing the severity of the injury. Studies in sheep have shown that CT (computed tomography) scan is potentially useful in assessing the severity of inhalational injury noninvasively. However, human trials are needed to establish the clinical utility of this method. A chest X-ray at presentation may be normal because radiographic changes often do not appear until 24 hours or more after the insult.

Management is primarily supportive. Early tracheal intubation, ventilatory support, aggressive pulmonary toilet, bronchodilator therapy, and bronchoscopic lavage all play an important part in the treatment of inhalation injury.

The ABC (airway, breathing, and circulation) approach should be used for managing patients with inhalational injury. There should be a low threshold for intubating patients with inhalational injury. Consider early intubation if there is stridor, Glasgow Coma Scale (GCS) < 8, hypoxia or hypercapnia, full thickness neck burns, deep facial burns, and oropharyngeal edema. A head-up position may delay swelling. With an abnormal airway or upper airway obstruction, the safest way to secure the airway is with the patient awake. Key prerequisites include effective topical anesthesia, proper patient positioning, and supplemental oxygen administration. Alert patients can be given intravenous (IV) opioid for pain relief, but sedatives should be used cautiously as it may worsen airway obstruction. The patient should be anesthetized using inhalational induction. Choice of intubation technique depends on the expertise of the operator. Fiberoptic intubation techniques are not useful in patients with swollen upper airways as it is impossible to anesthetize the airway and airway instrumentation may cause further trauma and swelling. Alternatives include direct laryngoscopy, the laryngeal mask airway, and use of Bullard laryngoscope. General

anesthesia with inhalational induction may be needed in uncooperative patients. Oral intubation should be done and cutting the tube should be avoided.

When the upper airway is badly damaged and endotracheal intubation is not possible, a direct surgical approach to the airway is indicated. Options include a needle cricothyroidotomy, surgical cricothyroidotomy, or tracheostomy; however, because of the high incidence of complications, a surgical airway should be considered only as a last resort.

Mild smoke inhalation can be managed supportively with high flow oxygen, humidified inspired gases, bronchodilators, and physiotherapy. Patients with more severe injury will need ventilator support for deteriorating lung function. FOB should be done in all intubated patients with suspected inhalational injury as it allows better diagnosis and enables aggressive pulmonary toilet.

Fluid restriction does not prevent pulmonary edema, however under-resuscitation will worsen the pulmonary damage. Ventilatory management should include the low tidal volumes (6 mL/kg) and low pressures (plateau pressures <30 cm H₂O) currently recommended for the management of the ARDS. Bronchodilators may be helpful in the treatment of bronchospasm following inhalation injury. Mucolytics such as N-acetylcysteine may help in the airway clearance of mucus plugs and may help in improving the PaO₂/FiO₂ ratios. Steroids and prophylactic antibiotics are not indicated. Management of CO poisoning and cyanide toxicity is discussed later.

Q. How would you resuscitate a patient with burns?

Ans. Treat according to cardiopulmonary resuscitation (CPR) protocol (ABCs). If there are any signs of airway obstruction or any suspicion of inhalation injury, then it is an indication for immediate endotracheal intubation. All burns patient should receive 100% oxygen with a reservoir mask.

First aid: In the initial first aid, one should try and stop the burning process and cool the burn wound. Use blankets to extinguish flame burns. Remove the heat source such as clothing, embers, and chemical. Synthetic fabrics burn at high temperatures and melt into a hot residue that continues to burn the patient. Chemical powders should be brushed from the wound. The burnt area should be rinsed with tepid water (at least 15°C for 20–30 minutes). This helps to limit the eventual depth and area of the resulting burn. Ice should never be used because it causes vasoconstriction leading to further tissue damage and hypothermia. The patient should then be covered with warm, clean, and dry linens to prevent hypothermia.

Primary Survey

- Airway maintenance with cervical spine control
- Breathing and ventilation
- Circulation with hemorrhage control
- Disability—neurological status
- Exposure and environmental control
- Fluid resuscitation proportional to burn size.

Airway and control of cervical spine: The first step is to identify inhalational injury in burns patient. A burns history can give an idea about the possibility of inhalational injury. Initial compromise of the airway is always due to low GCS. Coma may be due to trauma, drugs, and alcohol, as well as effects of CO and smoke inhalation. If there is any history of trauma, cervical spine should be immobilized. Airway can be supported with simple manual techniques or use of basic airway adjuncts. After clearing the airway, administer 100% oxygen via a reservoir mask; this would also help to treat CO poisoning. Elevate the head of bed to 30–90° to reduce the facial or airway edema provided there is no cervical spine injury. Endotracheal intubation and mechanical ventilation is needed in patients who are unconscious from coexisting trauma or from inhalation of toxic substances, patients who develop acute respiratory failure due to smoke inhalation and in patients with major burns.

Breathing: The chest should be exposed to ensure that chest expansion is adequate and equal. Look for signs of respiratory distress (increased respiratory rate, tracheal tug, and excessive use of accessory muscles). Circumferential burns to the chest may restrict breathing and impair gaseous exchange. An escharotomy may be required in circumferential burns that compromise breathing. Blast injuries related to the burn can cause lung contusions and alveolar trauma, potentially leading to ARDS. CO poisoning should be ruled out by measuring the carboxyhemoglobin (COHb) level. An arterial blood gas should be obtained to determine the COHb level. Pulse oximeter will give spuriously high readings in the presence of significant levels of COHb. High flow oxygen should be administered via a nonbreathing mask at 100%. Check for additional injuries that may have occurred when the patient sustained a burn injury (e.g., pneumothorax, hemothorax, tension pneumothorax, or a flail chest).

Circulation: Rapid assessment of the volume status should be done. Monitor the blood pressure (BP), pulse, and capillary refill in both burned and unburned limbs. Escharotomies may be required if there is isolated poor perfusion in a limb with circumferential full thickness burns. Use of a BP cuff may be difficult in a burned patient and invasive arterial monitoring may be needed. Stop any bleeding with direct pressure. If the burned patient is hypotensive then look out for other causes of hypotension.

Disability (neurological status): Establish the level of consciousness and examine the pupillary response to light. Restlessness and decreased level of consciousness may be due to hypoxemia and hypovolemia as well as concurrent head trauma. While taking history and assessing the airway, a simultaneous assessment of baseline GCS should be done in all patients.

Exposure and environment control: Remove any jewelry and clothing and keep the patient warm. Burns patients rapidly become hypothermic due to loss of their protective thermoregulatory skin and evaporation of fluid from the exposed tissue.

Fluid resuscitation: Following tissue burns, there is large fluid shift due to increased capillary permeability. This results in edema formation particularly during the first 36 hours. The resultant fluid depletion is greatest in the first few hours and early fluid resuscitation is essential to avoid hypovolemic shock and acute renal failure (ARF). Adults with >15% TBSA burns and children with >10% TBSA burns will need fluid resuscitation. While resuscitating burns patient, insert urinary catheter in order to monitor urine output as a function of organ perfusion. One should aim for a urine output of at least 0.5 mL/kg/h in adults and 1 mL/kg/h in children who weigh <30 kg.

After establishing airway patency and identifying and treating immediately life-threatening injuries, two large-bore IV cannulae should be inserted, preferably in a nonburned area. Upper extremities are preferable to lower extremities for venous access because of high incidence of phlebitis in the saphenous veins. Other options are saphenous cut down, femoral cut down, and intraosseous needles in children <6 years.

Warmed Ringer's lactate solution should be used for treating hypovolemic shock. Parkland formula is widely used for burns resuscitation. Fluid requirement is 4 mL/kg/% BSA of burns; give half in first 8 hours and the remaining over next 16 hours. In children, it is necessary to administer maintenance IV fluids containing glucose in addition to the burn formula. These formulae are only a guide and assessment of resuscitation should be based clinically on the heart rate, BP, capillary refill, central venous pressure (CVP), urine output, peripheral and core temperature, and mental state of the patient.

Blood should be collected for grouping and cross-matching, complete blood count (CBC), blood glucose, serum electrolytes, serum urea and creatinine, COHb levels, and arterial blood gas analysis. Urine should be tested for myoglobin/hemoglobin. Chest X-ray should be done. Other X-rays may be indicated if there are associated injuries.

Pain management: A severely burned patient may be restless and anxious due to hypoxemia and hypovolemia rather than pain. Hence, these patients respond better to oxygen or increased fluid administration rather than narcotic analgesics or sedatives which may mask the signs of hypoxemia or hypovolemia. Narcotic analgesics and sedatives should be administered in small frequent doses by IV route only. Intramuscular and subcutaneous injections should be avoided as absorption will be erratic and analgesia delayed.

Secondary Survey

Once the patient has been stabilized, he should be thoroughly examined for any concomitant injuries. After assessing the size and depth of burns, wash the burnt area and do the dressing. Use dressing (preferably transparent to facilitate wound inspection) to cover the burnt area as it protects the wound and reduces heat and evaporative losses.

Insert nasogastric tube and decompress the stomach if nausea and vomiting are present, in patients with burns >20% TBSA and all intubated patients. As patient with burns are in a highly catabolic state, establishing enteral feeding within 4 hours of admission helps to improve the outcome. Enteral feeding should be commenced as soon as possible after burn injury to promote normal gut function and to decrease the potential for bacterial translocation across the gastric mucosa.

Prophylactic antibiotics are not indicated in the early postburn period. Antibiotics should be reserved for treatment of infection.

Tetanus prophylaxis should be given.

Early referral to a burns center would be appropriate.

Q. What are the various formulas for fluid resuscitation in patients with burns? Which is the most commonly used formula?

Ans. The main aim of resuscitation is to support the perfusion of vital organs and also to maintain adequate tissue perfusion to the burn area itself and so prevent it from deepening. Historical overview of various burns resuscitation formulas is given in **Table 2**.

Parkland formula: It is the most widely used resuscitation formula in the world. Only the initial 24 hours crystalloid portion of the formula is followed closely with little attention focused on the subsequent 24 hours colloid recommendations. Children also require maintenance fluid at an hourly rate of 4 mL/kg for the first 10 kg + 2 mL/kg for the second 10 kg + 1 mL/kg for every kg thereafter. This can be given orally (preferably as a nutritious fluid such as milk), or intravenously using 5% dextrose and 0.45% normal saline solution, but not both together. The amount

TABLE 2: Formulas for fluid resuscitation.*Historical overview of various burn resuscitation formulas*

1942	Harkins formula	Any patient with at least 10% burn: Administer 1,000 mL plasma for each 10% TBSA burn over first 24 hours
1947	Body-weight Burn budget	First 24 hours: 1–4 L RL + 1,200 mL 0.5 NS + 7.5% body weight colloid + 1.5–5 L 5% D. Second 24 hours: Same formulation except change colloid to 2.5% body weight
1952	Evans formula	First 24 hours: NS at 1 mL/kg/% burn + colloids at 1 mL/kg/% burn + 2 L glucose in water Next 24 hours: Half of the first 24 hours crystalloid and colloid requirement + the same amount of glucose in water as in the first 24 hours
1953	Brooke formula	First 24 hours: RL at 1.5 mL/kg/% TBSA burn + colloid at 0.5 mL/kg/% TBSA burn Second 24 hours: 2 L 5% D
1968	Parkland formula	First 24 hours: RL at 4 mL/kg/% TBSA; give half in first 8 hours and the remaining over the next 16 hours Second 24 hours: Colloid at 20–60% of calculated plasma volume (55% of blood volume) to maintain adequate urinary output
1974	Muir and Barclay	Colloids (mL) = % TBSA burns × body weight in kg/2 Time blocks: 4 hourly for first 12 hours, 6 hourly for next 12 hours, after 12 hours. A total of 36 hours postinjury. Crystalloid: (1.5 mL × body weight) mL/h Blood: 50 mL/% BSA
1979	Modified Brooke	First 24 hours: RL at 2 mL/kg/% TBSA burn, one half in the first 8 hours and half in the remaining 16 hours Second 24 hours: Colloid at 0.3–0.5 mL/kg/% TBSA burn + 5% D to maintain urine output
1984	Monafo formula	First 24 hours: NS or RL to target urine output of 30 mL/h Second 24 hours: One-third of isotonic salt administered orally

(BSA: body surface area; D: dextrose; NS: normal saline; RL: Ringer's lactate; TBSA: total body surface area).

of fluid given should be constantly adjusted to maintain a urine output of 0.5–1 mL/kg/h in adults and 1–2 mL/kg/h in children. The fluids have to be calculated from the time of injury and not from the time of admission. The older fluid resuscitation formulas are confusing and difficult to recall. As a result of this, the United States Army Institute of Surgical Research (USAISR) has developed a simplified resuscitation formula named “The Rule of Ten” which is currently being implemented. It falls within American Burn Association (ABA) acceptable guidelines for fluid therapy. Initial fluid rate in mL/h = % TBSA × 10. For every 10 kg

above 80 kg, 100 mL is added to this rate. When compared directly to the Parkland formula, the fluid rate would be overestimated for patients under 40 kg and underestimated for patients over 140 kg. Further adjustments to the fluid rate can be made based on clinical observations of effect.

Q. What are the complications of fluid resuscitation?

Ans. Compartment syndrome: It occurs when the pressure within a compartment of the arm or leg or in the abdomen increases. Due to the increase in pressure, there is a decrease in blood supply resulting in damage to the muscles and nerves. This occurs with third spacing or extravasation of IV fluids. It also occurs with circumferential burns on the limbs or body. Children are at a higher risk for the compartment syndrome.

During resuscitation, careful monitoring should be maintained. Early recognition of development of compartmental syndrome is the key to management. Vigilant clinical monitoring is the most important factor for diagnosing any compartment syndrome. Limb compartment syndrome is treated by escharotomy or fasciotomy. Abdominal compartment syndrome is treated by surgical decompression and “leaving the abdomen open” (some form of protective dressing is used).

Q. How are burn wounds taken care of?

Ans. Typically, a burn wound has three layers. The center is the zone of coagulation where irreversible damage has occurred. Around this is the zone of stasis, characterized by impaired blood flow. This is the area which can be salvaged by timely resuscitation. The outermost is the zone of hyperemia due to vasodilation and recovery of tissue in this zone is usually good.

It is important to remove the necrotic tissue by debridement, as the necrotic tissue acts as a culture medium for bacterial growth giving rise to infections. The wound after debridement should be covered with antibacterial ointments and emulsifications to keep it moist and resistant to bacterial invasion. Many biological (potato peel, amniotic membrane, etc.) and commercial dressing materials are available. This also prevents fluid loss from the wound and helps in early recovery.

Q. What are the causes of CO poisoning?

Ans. Carbon monoxide is a colorless, odorless, toxic gas that is a product of incomplete combustion of carbon containing compounds such as wood, coal, and gasoline. The most common cause of fire-related deaths are due to smoke inhalation and CO poisoning. CO comes from both natural and manufactured sources. CO is produced in the human body as a by-product of hemoglobin degradation. Baseline

COHb saturation is 1–3% in nonsmokers and it increases to 10–15% in heavy smokers. Inhalation of combustible fumes produced by small gasoline engines, stoves, generators, lanterns, gas ranges, or by burning charcoal and wood in the household environment is the largest source of CO poisoning. Tobacco smoke, automotive exhaust (drivers stuck in traffic), and occupational or industrial sources (steel foundries and paper mills) can lead to chronic CO poisoning. Other sources could be furnaces, gas-powered engines, home water heaters, paint removers containing methylene chloride, pool heaters, and smoke from all types of fire. Exposure to any fossil fuel can result in acute CO poisoning.

Q. What is the mechanism of CO poisoning?

Ans. Carbon monoxide reduces the oxygen-carrying capacity of blood resulting in tissue hypoxia. The PaO_2 is normal. CO binds avidly to other heme-containing compounds, especially the cytochrome system. Possible mechanisms of toxicity include:

- Decrease in the oxygen-carrying capacity of blood
- Leftward shift of the oxyhemoglobin dissociation curve, further decreases oxygen delivery to the tissues
- Binding with cytochrome A_3 results in decrease in cellular respiration
- Binding to myoglobin, potentially causing myocardial and skeletal muscle dysfunction.

Incapacitation of the cellular mechanisms to utilize oxygen is one of the most important toxic action of CO which results in chemical asphyxiation and hypoxia.

Hemoglobin is a tetramer with four oxygen-binding sites. CO binds with the oxygen-binding sites as it has 250 times more affinity for hemoglobin than oxygen. As a result of this, oxygen cannot bind with the red cells which results in histotoxic hypoxia. COHb causes a leftward shift of oxyhemoglobin dissociation curve due to which oxygen is not readily released to the tissues. CO also competes with oxygen for other heme proteins, for example, myoglobin, peroxidases, catalases, and cytochromes. It also binds with cytochrome oxidase in the cells due to which cells are unable to utilize the oxygen.

CO binds to cardiac and skeletal myoglobin as well. CO binds to cardiac myoglobin three times more than skeletal myoglobin. Carboxymyoglobin dissociation is slower than COHb due to the increased affinity of CO for myoglobin. Fetal hemoglobin binds CO more avidly than hemoglobin A, and with slow transplacental transport, fetal levels decrease much more slowly than in the mother. This accounts for the occurrence of fetal death in nonfatal maternal exposures.

Q. What are the symptoms of CO poisoning?

Ans. To function normally, oxygen is required by the cells in the body; therefore, all body systems are affected by CO poisoning. The heart, brain, and lungs are most susceptible to the effects of CO poisoning as they need a continuous supply of large amounts of oxygen for normal function. Early signs and symptoms are due to the impairment of respiratory, cardiovascular, and neurologic systems. CO poisoning often mimics a viral illness with flu-like symptoms such as fatigue, lethargy, somnolence, malaise, nausea, vomiting, and dizziness. Headache, nausea, and dizziness are the most common symptoms. Cardiac contractility is decreased due to binding of CO to cardiac myoglobin which further decreases tissue oxygen delivery (low-flow hypoxia). Due to persistent changes in the redox state of cytochrome systems, the clinical signs of CO poisoning may persist long after blood COHb has returned to normal.

Various other clinical effects are seen due to widespread ischemia (rhabdomyolysis, pulmonary edema, multiorgan failure, disseminated intravascular coagulation, dermal injury, and renal failure). CO exposure can also be made worse by comorbidities such as congestive heart failure, anemia, drug ingestion, burns, trauma, and smoke inhalation.

The severity of symptoms depends on the COHb levels. Patient is usually asymptomatic if the levels are <10%. As COHb increases above 20%, the patient may develop headache, dizziness, confusion, and nausea. Coma and seizures due to cerebral edema are common with levels >40%, and death is likely above 60%.

COHb%	Symptoms
10%	Asymptomatic or may have headaches
20%	Dizziness, nausea and vomiting, and syncope
30%	Visual disturbances and fatigue
40%	Hallucinations and ataxia
50%	Seizures and coma
60%	Cardiopulmonary dysfunction and death

Q. How would you treat a patient with CO poisoning?

Ans. Acute CO poisoning is treated by terminating the exposure by removing the patient from the exposed environment and by administering supportive care. The binding of CO with hemoglobin is competitive and reversible. Hence, the mainstay of therapy for CO poisoning is supplemental O_2 , ventilatory support, and monitoring for cardiac arrhythmias. All patients should be given 15 L/min of oxygen via a nonrebreather mask. The half-life of COHb is 250 minutes at atmospheric pressure, 40–80 minutes while breathing 100% O_2 , and 22 minutes in a hyperbaric chamber at 2.5 atm. Oxygen therapy should be continued

because secondary peak of COHb occurs after 24 hours due to the washout of CO from the cytochrome.

Q. What is the role of hyperbaric oxygen (HBO) therapy in CO poisoning?

Ans.

- HBO therapy reduces the half-life of COHb to 20–30 minutes.
- It also increases the amount of oxygen dissolved in the blood from 0.3 mL/dL with isobaric therapy (FiO₂ 100%) to 5.5–6.4 mL/dL (2.4–2.8 atm).
- HBO induces cerebral vasoconstriction, which may reduce intracranial pressure and cerebral edema. HBO results in more rapid dissociation of CO from respiratory cytochromes.
- HBO may antagonize the oxidative injury that occurs after CO poisoning.

However, the precise role of HBO therapy is controversial and depends on the availability of hyperbaric facilities. Indications for considering HBO therapy would be:

- Any neurological abnormality or cognitive impairment
- Seizure
- Hypotension
- Chest pain, abnormal ECG (electrocardiogram) or elevated cardiac enzymes, and arrhythmias
- COHb levels >40%
- Pregnancy with COHb level >15%
- History of loss of consciousness or syncope
- Comatose patients.

Relative indications for HBO therapy include:

- Persistent neurologic symptoms (headache or dizziness) after 4 hours of high flow oxygen therapy via nonrebreathing mask
- Persistent acidosis
- Concurrent chemical or thermal burns
- Pregnancy with history of any CO exposure.

Q. Why do you get cyanide poisoning in burns patient?

Ans. Hydrogen cyanide is released as a product during the combustion of plastics and other polymers, silks, wool, cotton, and many other nitrogen-containing substances. Plastics and foams are increasingly being used in households as a result of which cyanide poisoning is likely in burns patients.

Q. What are the causes of cyanide poisoning?

Ans. Cyanides come from a wide range of natural and manmade sources. Both gaseous and solid forms of cyanide is used extensively, often in very large quantities, in industries including metallurgy, electroplating, and metal cleaning. Cyanide also is used in the recovery of gold and

silver from mineral ores and of silver from photographic materials; in the production of plastics, pigments, and dyes; and as a pesticide. Other sources of cyanide include emissions from the production of iron and steel, coal burning, vehicle exhaust, and cigarette smoke. Combustion of substances such as polyurethane, acrylonitrile, nylon, wool, and cotton can result in the production of hydrogen cyanide. Nitriles are used as solvents in the manufacturing of plastics. They can produce hydrogen cyanide when they are metabolized in the body after absorption through the skin or gastrointestinal tract. Some drugs [e.g., sodium nitroprusside, benzyl isothiocyanate, and amygdalin (a fraudulent cancer remedy)] and plant constituents (e.g., those in cassava or Puerto Rican lima beans) can generate cyanide in the body when they are metabolized.

Q. What is the pathophysiology of cyanide poisoning?

Ans. Cyanide causes tissue asphyxia by inhibiting intracellular cytochrome oxidase activity, the final step in oxidative phosphorylation, thus preventing mitochondrial oxygen consumption. Tricarboxylic acid cycle is also inhibited by cyanide poisoning. Affected cells can only generate adenosine triphosphate (ATP) via anaerobic metabolism. As a result, there is lactic acidosis due to anaerobic conversion of pyruvate to lactate. Cyanide poisoning should be suspected in a burns patient, if there is persistent lactic acidosis in spite of fluid resuscitation.

Cyanide poisoning can be difficult to diagnose. With a concentration of 50 parts per million (ppm), symptoms include headache, dizziness, tachycardia, and tachypnea. Above 100 ppm, lethargy, seizures, and respiratory failure occur. Possibility of cyanide poisoning should be suspected whenever there is a history of smoke inhalation. These patients have a high anion gap acidosis which does not respond to O₂ administration. Plasma lactate levels are useful alternative diagnostic tool as they correlate with cyanide levels. In patients with cyanide poisoning, the mixed venous O₂ saturation is high.

Cyanide is normally metabolized by hepatic rhodanese to thiocyanate, with thiosulfate as a substrate, but this process is slow. Administration of additional thiosulfate will speed up the hepatic metabolism of cyanide to produce nontoxic thiocyanate which is the excreted in urine. Nonhepatic metabolic pathways to remove cyanide include the combination of cyanide with methemoglobin and hydroxocobalamin. Administration of amyl sodium nitrite results in oxidation of hemoglobin to methemoglobin which combines with cyanide to form cyanomethemoglobin. Although methemoglobin does not transport oxygen, patients can tolerate methemoglobin levels of up to 40%, and this can be used to guide therapy.

Q. What are the problems with pulse oximetry in patients with burns?

Ans. Burns patients can have CO poisoning which in turn increases the COHb levels in the blood. Pulse oximeter may not be a reliable indicator of tissue oxygenation in these patients in the presence of COHb, as it overestimates the oxygen saturation. A co-oximeter, which measures the percentage of hemoglobin, oxyhemoglobin, COHb, and methemoglobin, is needed to obtain accurate oxygen saturation.

Q. What are anesthetic considerations for excision and grafting of major burn injuries?

Ans.

Preoperative Evaluation

History: Burns patient have a 48-hour period of initial stabilization after which they are posted for burn wound excision and skin grafting. A complete medical and surgical history should be taken and a thorough physical examination should be done. A burns history would tell you about the possibility of inhalational injury.

Examination: In the preanesthetic evaluation, the age and the percentage of TBSA of burns will give an idea about the patient's physiologic condition. The adequacy of resuscitation should be assessed. In the airway assessment, one should check the Mallampati class, thyromental distance, and head and neck mobility. The presence of facial burns can make mask ventilation difficult. Edema, scarring, or contracture formation may limit mouth opening and neck mobility. Traditional IV access sites may be unavailable.

Investigation: Blood grouping and cross-matching, CBC, blood glucose, serum electrolytes, serum urea and creatinine, and arterial blood gas analysis to know COHb levels should be done. Chest X-ray should be done. Adequate blood should be kept ready as blood loss has been estimated to range from 200 to 300 mL/% of body surface excised and grafted. ECG has to be done in patients above 40 years of age or if they have associated medical conditions.

Preoperative optimization/preparation: In major burns patients, it is extremely difficult to achieve adequate nutritional status as they are in a hypercatabolic state. Therefore, avoiding prolonged periods of fasting before the surgery would be beneficial. These patients are many times on analgesics for pain relief and should be continued through perioperative period. Depending on the extent of surgical excision, adequate-sized IV access should be taken.

Location of burns and donor skin sites indicate the need for special positioning, for repositioning the patient during operation, or both.

Monitoring: Intraoperative monitoring should include ECG, BP, end-tidal CO₂ (ETCO₂), pulse oximetry, core temperature, neuromuscular, and urine output monitoring. Cutaneous burns may make placement of conventional monitoring devices such as ECG electrodes or BP cuff difficult. Gel electrodes may not pick up ECG through damaged skin. Skin staples or subcutaneous needles attached to crocodile clips will give good signal. Invasive BP monitoring would be preferable in patients requiring extensive debridement. It may be difficult to get the pulse oximetry trace due to peripheral burns or vasoconstriction. Alternative sites such as ear, nose, or tongue can be used. COHb can give spurious results, hence arterial gas analysis with COHb levels should be done if there is any cause for concern. Because there is increased dead space in inhalation injury, ETCO₂ may not reflect PaCO₂. Arterial gas analysis should be used to monitor the ventilator settings. Central venous catheter may be needed in patients with extensive burns.

Temperature monitoring is essential because hypothermia is common and difficult to prevent. Patients with burns lose heat due to evaporation. Also, the cutaneous vessels in the burnt area cannot constrict and prevent heat loss by radiation.

The operating room ambient temperature should be 28–32°C and all topical and IV fluids should be warmed. When possible, nonoperative sites should be covered and forced air warming devices used.

Induction of anesthesia: General anesthesia with the combination of an opioid, muscle relaxant, and a volatile agent is the most widely used technique for burn excision and grafting. Laryngeal mask airway (LMA) has been shown to be effective in maintaining the airway in both adults and children with burns. Consider awake fiberoptic intubation if mouth opening is restricted. Tracheostomy is generally undesirable because of the risk that infection may spread to the damaged skin.

Various IV agents can be used successfully for induction. Ketamine has a lot of advantages. It not only provides stable hemodynamics, it also has analgesic properties. Hence, it has been used extensively as the primary agent for both general anesthesia and analgesia for burn dressing changes. Its major drawback is its tendency to produce dysphoric reactions. An antisialagogue premedication is a must. Diazepam or midazolam may be used to control the emergence hallucinations in some patients who receive ketamine. Etomidate can be an alternative to ketamine in hemodynamically unstable patient. Thiopentone can be used as an induction agent in patients who are adequately volume resuscitated and hemodynamically stable.

Succinylcholine should be avoided 24 hours after burns injury.

Maintenance of anesthesia: If laryngeal mask is used, the patient can be kept spontaneously breathing with O₂, nitrous, and inhalational agent. The choice of volatile agent does not appear to influence outcome from anesthesia for burn surgery; however, it must be kept in mind that these patients may require repeated anesthetics over a short period of time, hence the use of halothane should be avoided to reduce the risk of halothane hepatitis. If patient has been intubated, then nondepolarizing muscle relaxants (atracurium or vecuronium) can be used for controlled ventilation.

As these patients routinely receive opioids for various procedures or routine pain management, they develop tolerance and may need large doses of opioids in perioperative period for pain relief.

They have increased minute ventilation requirements due to increased metabolic rate and parenteral hyperalimentation. In patients with >40% burns, the metabolic rate is doubled, resulting in increased oxygen consumption and CO₂ production. Patients with ARDS due to inhalational injury may be PEEP dependent.

Regional anesthesia is useful in minor burns. However, in major burns, it is not very beneficial because skin may have to be harvested from extensive areas which may not be effectively covered by regional blockade. Also, regional techniques should not be performed through burned tissue because of the potential for infection to spread. Epidural local anesthetic should be used cautiously in extensive debridements as there is a potential for massive blood loss and hypotension. However, epidural opioids can be safely used.

Blood loss during excision and grafting procedures can be minimized by using topical epinephrine. Application of bandages soaked in 1:10,000 adrenaline after the excision of burned skin is effective in producing a bloodless surface for placement of skin grafts. After burn injury, cardiovascular responses to catecholamines are attenuated because of a reduced affinity of the β -adrenergic receptor for ligands and decreased second messenger production. This may explain the minimal changes in BP and heart rate observed after topical administration of such high concentrations of epinephrine.

Patients would need large amounts of fluids intraoperatively which can cause considerable soft tissue edema. If the patient is prone or if there is significant facial edema after the operation, then extubation should be delayed till the swelling subsides.

Postoperative Care

Patient-controlled analgesia (PCA) can be used in postoperative period using various opioids. Nonopioid analgesics such as nonsteroidal anti-inflammatory drugs (NSAIDs) or paracetamol can be considered depending on the presence or the absence of renal or liver dysfunction.

Q. Can suxamethonium be used for muscle relaxation in a patient with burns?

Ans. Suxamethonium can be used in the first 24 hours to facilitate endotracheal intubation. After burns, there is an increase in the number of postjunctional receptors which causes prolonged depolarization and marked release of potassium. This process of receptor proliferation takes several days to develop, with an initial window of safety of unknown duration. The most dangerous period is thought to be between 4 days and 10 weeks after thermal injury. How long the hyperkalemic response to suxamethonium persists is also unclear. However, it is advisable that suxamethonium should be avoided for up to 2 years postburns. It should also be avoided in patients with extensive muscle damage as it can cause the release of large amounts of potassium into the circulation sufficient to cause cardiac arrest. Rocuronium may be an alternative to suxamethonium.

Q. Discuss the problems with the use of nondepolarizing muscle relaxants (NDMR) in patients with burns.

Ans. Patients with thermal injury are resistant to the action of NDMR. This resistance develops by 1 week and usually persists for 8 weeks. Marked resistance to NDMRs only occurs when the burn is >30% TBSA. Proliferation of the acetylcholine receptors is responsible for the resistance to NDMR. The resistance to NDMRs implies that the burned patient will require larger than normal doses of NDMR to achieve a desired effect and that the duration of action will be shorter than normal. Hence, neuromuscular junction monitoring should be used to assess the adequacy of neuromuscular blockade as well as reversal with anticholinesterases.

Q. How would you give anesthesia for burns dressing?

Ans. This entails administration of anesthesia/analgesia away from the well-equipped operating room. IV ketamine and titrated doses of opiates are good options. It is necessary to ensure empty stomach. Limb burns dressing may be managed under epidural analgesia or peripheral nerve blocks as appropriate.

Q. A 25-years-old female patient with a history of burn injury to the face, hands, and upper part of the body 1 year back is posted for release of neck contracture. She has limited neck mobility due to contractures in the neck. She also has microstomia due to facial scarring and there

is severe scarring in both the upper limbs. How would you anesthetize this patient?

Ans. A complete medical and surgical history including a thorough examination of the patient would be required. Main concerns would be difficult airway in view of the limited neck mobility and microstomia. Due to the facial scarring, it may not be possible to get a good mask seal, making mask ventilation difficult. Insertion of an LMA would be difficult due to microstomia. Blind nasotracheal intubation is unlikely to succeed if the neck movements are restricted or it is fixed in flexed position. Microstomia and distortion of the oral cavity make use of the LMA and oral fiberoptic intubation difficult. Retrograde intubation may not be possible because of the extensive scarring on the anterior surface of the neck which distorts the surface anatomy of larynx. So, the technique of choice would be an awake fiberoptic intubation; however, one has to check for patency of external nares.

Intravenous access in the upper limbs will be difficult due to scarring. Since surgery does not require muscle relaxation, patient can be maintained on spontaneous ventilation using inhalational agents.

Q. What are the other options for release of neck contracture?

Ans. Tumescence local anesthesia can be used for release of neck contracture. It is a type of infiltration anesthesia where a large volume of local anesthetic with adrenaline is infiltrated into the subcutaneous tissue and after a waiting period of 20 minutes, the neck contracture can be released. Some surgeons use it as a sole means of anesthesia. One formula uses 25 mL of 2% lidocaine and 1 mL of 1:1,000 adrenaline for each liter of sodium lactate IV infusion. Dose of the local anesthetic depends on the site and indication and large volumes ranging from 70 to 200 mL may be required depending on the extent of contracture. Lignocaine doses of up to 35 mg/kg have been used without any side effects. This is because the dilution of lidocaine and epinephrine-induced vasoconstriction diminishes and delays the peak plasma lidocaine concentrations, thereby reducing potential toxicity. Infiltrating a large volume of dilute epinephrine assures diffusion throughout the entire targeted area while avoiding tachycardia and hypertension. Clinical local anesthesia can last as long as 18 hours, obviating the need for postoperative analgesia. If required, analgesic doses of ketamine 0.5 mg/kg can be given. In children, anesthetic doses of ketamine would have to be given at the beginning and the surgery can be carried out under ketamine and tumescence anesthesia. Ketamine is preferred over thiopentone or propofol because it preserves the hypoxic ventilation responses and airway reflexes. O₂ can be supplemented with nasal prongs or face mask.

Q. How is the pharmacology of drugs affected in burns?

Ans. Pharmacokinetic effects: The pathophysiologic changes occurring after thermal injury alter pharmacokinetic parameters such as absorption, bioavailability, protein binding, volume of distribution, and clearance. The extent of these changes depends on the magnitude of injury and the time between injury and drug administration.

In the acute phase after a burn, organ blood flow is reduced because of hypovolemia and decreased cardiac output. Drugs administered by routes other than IV route are likely to show delayed absorption.

Plasma albumin concentrations decrease and α_1 -acid glycoprotein levels increase. Plasma protein binding of albumin-bound drugs such as benzodiazepines is decreased, resulting in an increase in the free fraction and thus a larger volume of distribution for the drug. Raised fibrinogen and α_1 -acid glycoprotein will reduce the free fraction of basic drugs (local anesthetics, propranolol, and muscle relaxants). The changes in the nonprotein bound fraction of the drug can alter the pharmacological response.

However, because most anesthetic drugs are not highly protein bound, and because the hemodynamic changes with burns are so marked, the effect of protein binding on the pharmacologic effects of anesthetics is minimal. In addition, fluid loss to the burn wound and edema elsewhere can decrease plasma concentrations of many drugs below those expected in the unburned patient.

After the initial resuscitation phase, cardiac output increases as the hypermetabolic state develops. This increases blood flow to the kidneys and liver with increased drug clearance. However, there is wide patient-to-patient variability in renal and hepatic function after burns, so drug therapy must be tailored to each patient.

The volume of distribution of the drug may be altered by changes in protein binding and in extracellular fluid volume. Changes in the loading dose of the drug may be required if the drug has a small volume of distribution or a narrow therapeutic range. Total plasma clearance must be considered when considering drug maintenance doses and dosage intervals.

Pharmacodynamic changes: Changes in the drug-receptor interaction are common after burns and appear to account for many of the clinically important alterations in anesthetic pharmacology.

Burn injury causes proliferation of the extra-junctional receptors leading to resistance to nondepolarizing muscle relaxants and hypersensitivity to depolarizing muscle relaxants. This effect may occur within a week of injury, persists for up to a year, and is proportional to the total burns surface area.

Dose requirements for all anesthetic agents are increased due to hyperdynamic circulation and hypermetabolic state. Minimum alveolar concentration (MAC) values are increased and duration of action is decreased.

These patients also develop tolerance to the effects of sedatives, analgesics, and inotropic medications.

Q. What is the mechanism of burn pain?

Ans. The skin consists of three layers, namely the outer epidermis, inner dermis, and hypodermis. The sensory structures are contained within the dermis and consist of free nerve endings (for pain, temperature, and touch), Meissner's corpuscles (light discriminatory touch), and Pacinian corpuscles (pressure). Fibers from the free nerve endings travel in A δ fibers (fast pain) and unmyelinated C fibers ("slow" and chronic pain) to synapse in the substantia gelatinosa of the dorsal horn of the spinal cord. Fibers then cross the midline of the spinal cord to ascend to the thalamus in the lateral spinothalamic tracts and from there to the postcentral gyrus, where conscious perception of the stimulus may occur.

The instant pain that follows a burn injury is due to stimulation of skin nociceptors that respond to heat (thermoreceptors) and mechanical distortion (mechanoreceptors). Exogenous (e.g., hydrofluoric acid) and endogenous (e.g., inflammatory mediators, notably histamine, serotonin, bradykinin, leukotrienes, and prostaglandins) chemical stimuli are also responsible for pain after burn injury. Nerve endings that are entirely destroyed will not transmit pain, but those that remain undamaged and exposed will generate pain. The immediate pain sensations are carried by the unmyelinated C and thinly myelinated A δ fibers.

Subsequently, there is primary and secondary hyperalgesia.

Primary hyperalgesia: Following burns, there is a massive inflammatory response resulting in the release of inflammatory mediators which sensitizes the active nociceptors at the site of injury. Due to this, the wound and the skin immediately adjacent to it become sensitive to mechanical stimuli such as touch, rubbing, or debridement, and chemical stimuli such as antiseptics or other topical applications.

Secondary hyperalgesia: It is the increased sensitivity in the surrounding unburned areas resulting from continuous or repeated peripheral stimulation of nociceptive afferent fibers. This is mediated by the spinal cord and exacerbated

by the mechanical stimulation that occurs as a result of frequent dressings changes.

Different sizes and degrees of burn can result in differing amounts of pain. Usually, the larger the burnt area, the more the pain and the deeper the burn for a given area, the less the pain (because there is greater destruction of nerve endings).

Q. Discuss pain management in patients with burns. What analgesics are used?

Ans. Pain management is a problem in these patients. IV morphine remains the gold standard for the management of pain in patients with burns who are not at a risk of airway obstruction. These patients often need multiple dressing changes and various operations as a result of which they develop a tolerance to opioids very rapidly and therefore may require large doses of opioids and use of other analgesics.

In the initial first aid, cooling with tepid water reduces the pain. On admission to the hospital, pain can be treated with titrated doses of IV morphine. NSAIDs can be used in patients with minor burns. They provide pain relief by inhibiting the formation of inflammatory mediators such as prostaglandins released during burns. However, NSAIDs should be used with caution in patients with shock.

For postoperative analgesia, multimodal analgesic technique combining paracetamol, NSAIDs, local anesthesia, and opioids (IV morphine PCA) can be used. Local anesthetic solutions can be directly applied to skin donor sites. Although there is a theoretical risk of local anesthetic toxicity following unpredictable absorption over a large area, studies have not demonstrated this to date.

Regional blocks may be used but there may be practical difficulties such as infection close to the site of insertion, generalized sepsis, and coagulation abnormalities.

Q. How would you manage chronic pain after burn injury?

Ans. Neuropathic pain develops secondary to nerve damage, abnormalities in nerve regeneration and central nervous system reprogramming. This can destroy sleep pattern, result in depression, and impair rehabilitation. There may be hyperalgesia (increased response to painful stimulus) and allodynia (painful response to normally innocuous stimulus) which can start early in the postinjury course and persist for many years after the initial injury. The severity of pain is related to the size of burn and the number of skin grafts performed.

This type of pain is very difficult to treat with conventional analgesics. Treatment includes antidepressants (amitriptyline), anticonvulsants (gabapentin and sodium

valproate), regional nerve blocks, and cognitive behavioral therapy.

Q. How are electrical burns different from thermal burns?

Ans. Low voltage causes local contact burn. Domestic supply is 50 Hz alternating current (AC), 240 V which can cause cardiac arrest. Muscle spasm may prevent release of the electrical source. There is no associated deep tissue damage.

High voltage (>500 V) causes flash burns or deep tissue damage due to current transmission. When electric current comes in contact with the patient's body, the body acts as a conductor. The conversion of high-voltage electric energy to thermal energy results in burns. The amount of thermal energy transferred depends on the voltage of the electrical source, skin resistance of the victim, and duration of contact with the source of electrical current. The entry and exit burns are usually very small; however, there is massive underlying muscle damage. This is because the deeper tissues cannot dissipate the thermal energy as rapidly as more superficial tissues.

Patients with electrical burns need continuous ECG monitoring for at least the first 48 hours after burns as cardiac dysrhythmias are very common.

Respiratory arrest can also occur and may be due to respiratory muscle paralysis or tetanic contractions or some indirect trauma postinjury.

Rhabdomyolysis can cause release of myoglobin which can cause ARF. These patients should receive IV fluids to maintain a urine output of at least 1.5–2 mL/kg/h. Mannitol is recommended to preserve renal function once adequate fluid resuscitation has been achieved.

The affected limbs should be observed carefully as compartment syndrome may develop.

Neurologic complications are very common. They may result from direct injury or may occur later due to perineural scarring or neural ischemia.

Cataract formation is another sequel of electrical burn injury.

Q. What indicates high risk for arrhythmias after electrical injury?

Ans. The high-risk criteria for arrhythmias after electrical injury are as follows:

- Abnormal ECG on presentation
- Loss of consciousness at the time of electrical injury
- Exposure to high voltage (>240 V)
- Past cardiac history
- Unwitnessed event
- Increased skin conduction, e.g., wet skin and high humidity
- Tetany at time of electrocution.

Q. What is lightning electrical burn?

Ans. Lightning is an electrical burn of extremely high voltage (approximately 10–120 million volts). Lightning strikes are rare and only 3–5% of these are direct injuries. Most of the times the lightning strikes inanimate objects such as the ground, tree, or other objects that, once hit, transmit this energy to the victim.

Type of injuries: Only one-third of injured people have signs of burns and they too are usually superficial. The injuries are primarily neurological and affect all three components of the nervous system: central, autonomic, and peripheral. Cardiac and respiratory arrests may also occur. Indirect injuries from falling, being thrown by muscle contractions, or barotrauma from the explosive force of a nearby lightning strike may occur.

Q. How is chemical burn different from a flame burn?

Chemical burns can result from exposure to acids, alkalies, or petroleum products. Industrial or household alkalis and acids are commonly used chemicals, e.g., bleach, washing powder, disinfectants, and drain cleaner. Immersion in complex hydrocarbons (petrol and diesel) without ignition may cause systemic toxicity. Phosphorous burns can result from fireworks and military applications.

Hands and upper limbs are most frequently affected areas. Management is same as for flame burns with the exception that the burnt area should be irrigated with water for several hours. Irrigation helps to fully remove the corrosive substance because residual chemicals on the skin and clothing will allow the burning to continue.

Chemical burns are influenced by duration of contact, concentration of chemical, and amount of the agent. Alkali burns are more serious than acid burns because alkalies penetrate more deeply. Contaminated clothing should be removed at the earliest. The chemical should be immediately flushed with large amounts of water using a shower or hose, if available. Any dry powder on the skin should be brushed away before irrigation with water. Irrigation should be carried out for at least 20–30 minutes. Alkali burns to the eye requires continuous irrigation during first 8 hours after burns. Irrigation should be continued till the pain stops.

Neutralizing agents do not have any advantage over water lavage, because reaction with the neutralizing agent may itself produce heat and cause further tissue damage.

Hydrofluoric acid used for etching in glass industry is highly toxic. Burns of 2% TBSA can be fatal as it can cause significant and life-threatening systemic hypocalcemia. First remove contaminated clothing and irrigate with water. Treatment includes topical 2.5% calcium gluconate burn gel.

SUGGESTED READING

1. Advanced Trauma Life Support Manual. American College of Surgeons, 7th edition, 2004. Focus on: Burns Care, Current Anaesthesia and Critical Care. 2008;19(5-6):255-360.
2. Alvarado R, Chung KK, Cancio LC, Wolf SE. Burn resuscitation. *Burns*. 2009;35(1):4-14.
3. Black RG, Kinsella J. Anaesthetic management for burns patient. *BJA CEPD Reviews*. 2001;1(6):177-80.
4. Cancio LC, Batchinsky AI, Dubick MA, Park MS, Black IH, Gómez R, et al. Inhalation injury: pathophysiology and clinical care proceedings of a symposium conducted at the Trauma Institute of San Antonio, San Antonio, TX, USA on 28 March 2006. *Burns*. 2007;33(6):681-92.
5. MacLennan N, Heimbach DM, Cullen BF. Anesthesia for major thermal injury. *Anesthesiology*. 1998;89(3):749-70.
6. Mlcak RP, Suman OE, Herndon DN. Respiratory management of inhalation injury. *Burns*. 2007;33(1):2-13.
7. Norman AT. Pain in the patient with burns. *BJA CEPD Reviews*. 2004;4(2):57-61.
8. Walton JJ, Manara AR. Burns and smoke inhalation. *Anaesth Intens Care Med*. 2005; 6(9):317-21.

Anesthesia for Cleft Lip and Palate Surgery

Vandana Agarwal, Aparna Chatterjee

A 2-year-old child comes with history of nasal regurgitation of fluid and food since birth. The patient has a nasal twang, delayed development of speech, and recurrent respiratory infections. The child was full term at birth and does not have any other significant medical history. This patient is posted for cleft lip and palate (CLP) surgery.

Cleft lip and palate is one of the most common congenital malformations, associated with difficulty in feeding, speech development, and facial disfigurement. Surgery not only restores the function but also results in the defects being unrecognizable. Depending on the extent of the defect, different surgical manipulations may be required in various stages such as early orthopedic manipulation of the dental arch followed by cleft lip (CL) repair, palate repair surgery for velopharyngeal incompetence, and maxillary advancement at a later stage. These cases pose a challenge to the anesthetist as they present at a very young age, may have associated congenital anomalies, varying degrees of difficult airway, repeated corrective surgeries, and lastly the airway is shared with the surgeons.

Q. What are the types of cleft lip (CL)?

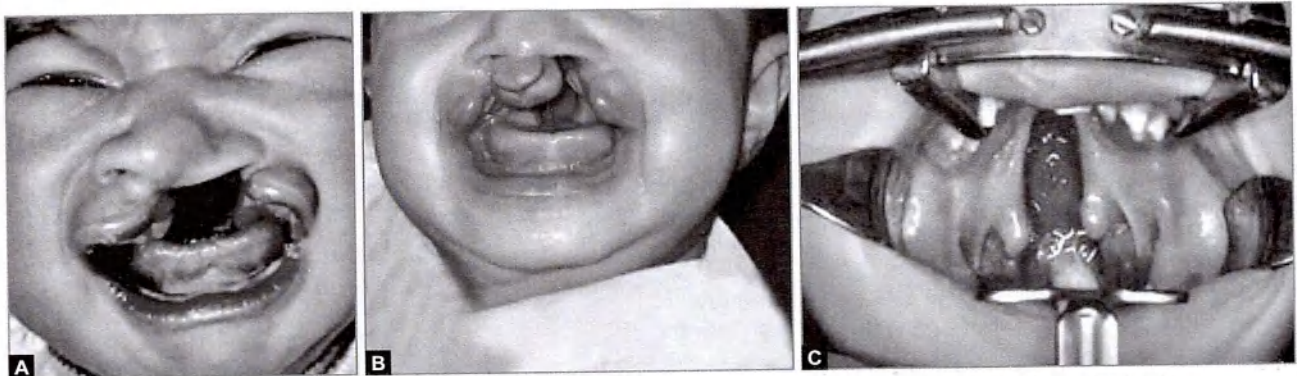
Ans. The CL can be classified anatomically by inspection.

- *The cleft can be unilateral or bilateral:* Bilateral clefts are frequently associated with a deficiency of central columella and elongation of the vomer, which causes protrusion of the anterior aspect of the premaxillary process. In some cases, the premaxilla is completely absent.
- *It can be complete or incomplete:*
 - *Complete CL:* Involves the entire lip going into the nostril

- *Incomplete CL:* Ranges from small to large defects. The cleft may be associated with nasal deformity, absence of nasal floor, and deformity of ala nasi.

Q. What are the types of cleft palate (CP)?

Ans. Cleft palate can be unilateral or bilateral fissure within the soft palate which may extend into the hard palate. CP may occur along with CL when the lip defect extends beyond the incisive foramen and includes the sutura palatina. Defects anterior to the incisive foramen are primary (prepalatal) clefts and posteriorly are secondary (postpalatal) clefts.



Figs. 1A to C: Children with cleft lip and palate (CLP): (A) Complete left-sided CLP; (B) Premaxilla; (C) Post-palatal cleft.

- The primary CP (prepalatal) involves anterior palate, alveolus, lip, nostril floor, and ala nasi. They can be complete or incomplete.
- The secondary CP (postpalatal cleft) is posterior to incisive foramen. They can be complete or incomplete depending on whether or not they extend all the way through the soft and hard palates to the incisive foramen.
- The last type of palatal cleft is the submucosal cleft in which a bone defect exists without a mucosal defect.

The most common cleft of the palate is a left complete cleft of prepalatal and postpalatal structures. The second most common is midline cleft involving the entire soft palate and part of the hard palate without involving the prepalatal structures (Figs. 1A to C).

Embryology

During the first trimester, defects in the palatal growth leads to cleft lip and palate (CLP). The palate grows inward to fuse in the midline in two stages. Initially by about 6 weeks, the primary palate which forms the alveolus and lip fuses weeks later to be followed by the secondary palate which is posterior to the incisive foramen. The suggested theories for failure of fusion include mechanical obstruction by tongue position, structural hypoplasia, or primary breakdown.

Q. What are the pathophysiological features of CLP?

Ans.

- CLP results in difficulty in feeding since birth. Suckling and swallowing is impaired. The neonate/infant may fatigue during feeding leading to inadequate weight gain and nutritional deficiencies and anemia. Recent literature suggests that perioperative complications do not increase with mild degrees of anemia (Hb 8–10 g/dL).
- Chronic upper respiratory tract infections are common in these patients secondary to regurgitation of fluids and food predisposing them to lower respiratory tract and ear infections. If the child at the time of surgery has acute upper respiratory tract infection, it is wise to postpone the surgery for 2–3 weeks as the postoperative respiratory complications increase particularly with general endotracheal anesthesia.
- CLP may be associated with other congenital anomalies/syndromes which may have their own symptomatology (see next question for details).
- CP leads to problems with phonation of the palatal consonants such as P, R, T, and D.

Q. What other problems are associated with CLP?

Ans. Up to 70% of patients with CLP have an isolated anomaly and are not associated with any syndrome. However, isolated CP may be associated with syndromes (Table 1 for some of the syndromes).

TABLE 1: Syndromes associated with cleft lip and palate and their anesthetic implications.

Syndrome	Associated anomalies	Anesthetic implications
Pierre Robin sequence	• 80% associated with cleft palate • Micrognathia • Glossoptosis • Underlying syndrome/anomalies	• Difficult airway • Postoperative respiratory obstruction • Usually easier to intubate with increasing age
Treacher Collins syndrome	• 28% associated with cleft palate • Micrognathia and maxillary hypoplasia • Choanal atresia • Eye and ear malformations	• Intubation may become more difficult with age • Postoperative respiratory obstruction
Hemifacial microsomia (Goldenhar syndrome)	• Hemifacial and mandibular hypoplasia • Cervical spine abnormalities • Ear and eye abnormalities	• Intubation may become more difficult with age
Velocardiofacial syndrome	• Microcephaly and microstomia • Flat nasal bridge, small ears, short stature, immune deficiency, and congenital cardiac disease • Laryngeal and tracheal anomalies • Velopharyngeal incompetence with/without cleft palate 22q 11 deletion (FISH test)	• Difficult airway • Antibiotics for congenital heart disease
Stickler syndrome	• Progressive connective tissue disorder • Micrognathia and flat face • Eye, ear, and joint abnormalities • Congenital cardiac disease	• Difficult airway—both mask ventilation and intubation can be very difficult
Down syndrome	• Microstomia and relative macroglossia • Epicanthic folds and simian crease • Congenital cardiac disease • Atlantoaxial subluxation and instability	• Difficult intubation • Caution with neck positioning • Antibiotics for congenital heart disease • Prone to airway obstruction

Contd...

Contd...

Syndrome	Associated anomalies	Anesthetic implications
Fetal alcohol syndrome	• Smooth philtrum and thin vermillion • Small palpebral fissures • Growth retardation • CNS abnormalities	• Difficult airway • Antibiotics for congenital heart disease
Klippel-Feil syndrome	• 15% associated with cleft palate • Short, webbed neck, and fused cervical vertebrae • Congenital cardiac disease • Renal anomalies • Scoliosis	• Difficult intubation • Antibiotics for congenital heart disease

(CNS: central nervous system; FISH: fluorescence in situ hybridization).

Q. What is VATER or VACTERL association?

Ans.

- V—vertebral anomalies
- A—anal atresia
- C—cardiovascular anomalies
- T—tracheoesophageal fistula
- E—esophageal atresia
- R—renal (kidney) and/or radial anomalies
- L—limb anomalies (in front of or above the central axis of the limb).

Q. When is the appropriate time to undergo surgery?

Ans.

- The common time for surgical repair of a CL is 3 months after birth; however, isolated CL without any other associated conditions can be closed as early as the first week of life. This makes suckling easier.
- The hard palate can be repaired up to 4–5 years of age in various stages but the soft palate should be closed prior to speech development, i.e., between 12 and 15 months of age.
- These children may require a secondary procedure in later years for cosmetic purposes. If child has a CLP, the lip is repaired first because the repaired lip helps to decrease the width of palate defect.

Fetal surgery to correct the CL with minimal or no scarring is in the experimental stages.

Q. What is Treacher Collins syndrome?

Ans. Treacher Collins syndrome, also known as mandibulofacial dysostosis, causes bilateral and symmetric deformities of head and face (**Figs. 2A and B**). It is an autosomal dominant disorder and has a prevalence of 1 case per 25,000 to 1 case per 50,000 live births. Both sexes are equally affected. It occurs due to genetic mutation in chromosome 5, which affects facial development.

Characteristics include:

- Palpebral fissures—slanting downward
- Notched lower eyelids
- Underdevelopment or absence of maxilla and lateral wall and floor of the orbit
- Retrognathia and posteriorly positioned tongue (creating an impression of a large tongue due to reduced pharyngeal space)—difficult airway and predisposing to postoperative airway obstruction. Relief of obstruction may need lateral or prone positioning to maintain airway. Rarely, a tracheostomy may be needed
- Ear abnormalities—hearing loss
- CP in one-third of the patients, palatopharyngeal incompetence may be present in additional one-third of the patients leading to difficulty in feeding and swallowing.

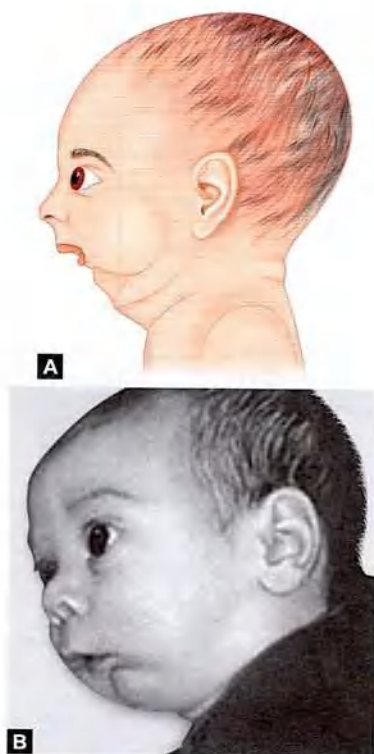


Figs. 2A and B: Treacher Collins syndrome. (A) Line diagram; (B) Clinical photograph.

Q. What is difference between Pierre Robin sequence and syndrome?

Ans. A “syndrome” describes errors of morphogenesis with simultaneous presence of multiple anomalies caused by single etiology, whereas the term “sequence” describes a clinical pattern (a series of anomalies), initiated by a single anomaly, in this case micrognathia.

The triad of micrognathia, CP, and retroglossoptosis was known as *Pierre Robin syndrome* until 1974 (**Figs. 3A and B**). The term *sequence* was recently introduced to include any condition that consists of a sequence of events initiated by a single malformation. It has a prevalence of approximately 1 case per 8,500 live births. Equally affects both sexes, except in the X-linked form. An autosomal recessive inheritance is possible.



Figs. 3A and B: Pierre Robin syndrome. (A) Line diagram; (B) Clinical photograph.

Various theories exist to explain the pathogenesis of Pierre Robin sequence. The most accepted theory is the mechanical theory. The initiating event is mandibular hypoplasia which keeps the tongue high within the oral cavity; this in turn causes a CP by preventing the closure of the palatal shelves. Alternatively, mandibular growth may be restricted mechanically in utero by oligohydramnios or due to abnormal development of the temporomandibular joint.

Characteristics include:

- Micrognathia
- Glossoptosis may cause severe breathing and feeding difficulty in the newborn. Also, obstructive sleep apnea may occur
- CP
- Otitis media occurs most commonly. There may be conductive hearing loss

- Speech development may be affected. Velopharyngeal insufficiency is usually more pronounced in these patients than in those with isolated cleft of the lip
- Cardiac defects may include benign murmurs, pulmonary stenosis, patent ductus arteriosus, patent foramen ovale, and atrial septal defect
- Musculoskeletal anomalies
- Other associated syndromes and conditions include Stickler syndrome, trisomy 18 syndrome, and velocardiofacial syndrome.

When managing a patient with CLP, even the ones diagnosed as “Pierre Robin syndrome or sequence,” it is essential to have a high index of suspicion for other underlying problems or syndromes.

Q. What are the anesthetic considerations for this child with left-sided CLP undergoing surgery?

Ans. Key concerns dominating the preoperative assessment of an infant posted for CLP surgery are:

- Pediatric patient
- Assessment and evaluation of associated congenital birth defects or a syndrome, if present
- Possibility of a difficult intubation
- Shared airway.

Preoperative Assessment

History

In addition to the usual information elicited (developmental milestones and vaccination history) during the preoperative interview, particular attention should be given to the following:

- Patients with a CLP are at risk of recurrent upper airway and ear infection secondary to regurgitation of fluid and food through the cleft; it is essential to rule out any upper respiratory infection to avoid postoperative complications. The risk of postoperative respiratory tract infection increases particularly in patients with greater severity of defect (e.g., a child with bilateral CLP has a significantly higher risk compared to another with unilateral CLP, 9% vs. 2% respectively) and requiring general anesthesia. In some cases, it is advisable to defer the surgery for 2–3 weeks until symptoms improve and the airway reactivity decreases. It is also important to note that following surgery, the risks of postoperative respiratory tract infection decreases, so the case should be individually assessed regarding the risks of postoperative respiratory infection and benefits of surgery.
- These children may have associated congenital heart defects (ASD, VSD, and mitral valve prolapse, some may have cyanotic congenital heart disease). Most of them would have undergone cardiology evaluation, and necessary information can be sought. If the child has not

undergone cardiology evaluation, a thorough evaluation of the cardiovascular system is essential.

- If there are associated syndromes, the pathophysiology of the syndromes should be thoroughly understood before administering anesthesia.
- Enquire about breathing or feeding difficulties since birth, how they were managed, and whether they have resolved.

Examination

- They should be examined for signs of retrognathia.
- The methods to evaluate airway in adults are not useful or have several limitations when applied to these cases. The infant/child may not cooperate, no specific thyromental distance or any other tests have been validated as a predictor for difficult intubation.

Investigations

- *Full blood count*: To look for anemia and evidence of infection.
- *Echocardiogram*: If there is a murmur detected on examination or if there is associated anomaly to assess the structure of the heart.

Preoperative Preparation

- Blood should be grouped and cross-matched as palatal surgery may bleed, particularly so in patients with anemia.
- Antibiotic prophylaxis is administered if there is coexisting congenital heart disease.
- Sedative premedication should be avoided if difficult airway is suspected or anticipated. Premedication with oral atropine may be done to decrease secretions and the possibility of laryngeal spasm. Atropine administered orally in a dose of 0.02 mg/kg (maximum dose 0.6 mg) 45 minutes prior to induction is effective.
- Pediatric difficult airway kit should be made available, if anticipating difficult airway.

Anesthetic Management

Induction of Anesthesia

- Monitoring include electrocardiogram (ECG), noninvasive blood pressure (NIBP), pulse oximetry, end-tidal capnography, temperature, and precordial stethoscope. Inhalational induction with sevoflurane is the choice in patients with anticipated difficult airway. Nitrous oxide may be administered in addition to sevoflurane to speed the induction.
- In patients without any airway problems/difficulty, intravenous induction with either thiopentone or propofol, fentanyl, and muscle relaxant can be done.
- During laryngoscopy if a large cleft is present on the left, a lubricated dental roll or gauze can be placed within

the gap to prevent trauma to the underlying tissue. Alternatively, the laryngoscope can be used to approach from the side rather than the midline.

- The trachea is preferably intubated with preformed oral endotracheal tube [Ring-Adair-Elwyn (RAE) tube and South Pole tube]. Alternatively, an Oxford tube can be used. The endotracheal tube rests in the midline on the tongue underneath the tongue plate of the Dingman mouth gag. It is secured in the midline over the chin.
- A throat pack is usually inserted by the surgeon to prevent aspiration of blood.
- Antibiotic prophylaxis against *Staphylococcus aureus* should be given following induction.
- The endotracheal tube position should be checked for any misplacement and obstruction after the patient is positioned and following the insertion and opening of a mouth gag. The ventilation should be monitored carefully during the placement of the mouth gag for any compression or displacement leading to increased airway pressure or leak.
- Once the airway is secured, ventilation is controlled to maintain normocapnea.
- Surgeons will often use adrenaline infiltration to reduce bleeding, the anesthetist must make sure that the concentration used in 1:200,000 and the appropriate amount is used (rule of the thumb 1 mL/kg).

Maintenance of Anesthesia

- Anesthesia can be maintained with nitrous oxide, inhalational agent such as isoflurane, fentanyl, and muscle relaxant such as vecuronium or atracurium.
- Blood loss should be assessed and blood should be transfused, if necessary.
- Fluid warmer and convective warming blanket should be used to prevent hypothermia.
- At the end of the procedure, the throat pack should be removed and oral cavity should be examined for swelling of the tongue, uvula, and any evidence of bleeding. A gentle laryngoscopic examination and suction should be done. If there is any evidence of swelling of the tongue or uvula, extubation should be postponed and these patients should be electively ventilated till the swelling resolves, especially following a prolonged surgery.
- Postoperative analgesia can be achieved by using infraorbital nerve block and administering systemic analgesics, before the effect of block wears off. Diclofenac suppository (1 mg/kg, 12 hourly) is commonly used. A loading dose of paracetamol 30 mg/kg rectally may be administered postinduction. Infraorbital nerve block can be administered following induction of anesthesia or after CL surgery.

- Neuromuscular blockade should be reversed with neostigmine and glycopyrrolate.
- The child should be extubated when awake and has full control of airway reflexes.

Postoperative Care

In patients with CP following surgery the size of the airway decreases, this can predispose them to postoperative airway obstruction at the time of extubation.

Q. What are the causes of postoperative airway obstruction?

Ans. Causes include:

- After palatal surgery, reduction in the size of the airway
- Edema of the tongue due to pressure from the mouth gag
- Residual effect of anesthesia and/or opioids
- Laryngeal edema following endotracheal intubation.

All patients at risk of delayed airway obstruction should be monitored closely in the postoperative period.

Q. What are the preoperative fasting guidelines for pediatric patients?

Ans. Children can safely be allowed clear fluids 2 hours before surgery without increasing the risk of aspiration. Food should normally be withheld for 6 hours prior to surgery in children aged 6 months or older. In children under 6 months of age, it is probably safe to allow breast milk but not formula or nonhuman milk up to 4 hours before surgery (Table 2).

TABLE 2: The fasting guidelines of American Society of Anesthesiologists.

Clear liquids (e.g., include but are not limited to water, fruit juices without pulp, carbonated beverages, clear tea, and black coffee. Does not include alcohol)	2 hours
Breast milk	4 hours
Infant formula	6 hours
Nonhuman milk	6 hours
Light meal (toast without butter and clear liquid)	6 hours
Full meal (fried/fatty meal)	≥8 hours

Q. How do you replace perioperative fluids in pediatric patients?

Ans.

- Hypovolemia should be corrected rapidly to maintain cardiac output and organ perfusion.
- In a child, the fall in blood pressure is a late sign of hypovolemia.
- Maintenance fluid requirements should be calculated using the Holliday-Segar formula:
 - *Body weight:* Daily fluid requirement
 - 0–10 kg: 4 mL/kg/h
 - 10–20 kg: 40 mL/h + 2 mL/kg/h above 10 kg
 - >20 kg: 60 mL/h + 1 mL/kg/h above 20 kg.

- A fluid management plan for any child should address three key issues:

- Any fluid deficit which is present
- Maintenance fluid requirements
- Any losses due to surgery, e.g., blood loss and third space losses.

During surgery, all these requirements should be managed by giving isotonic fluid, such as Ringer's lactate/Hartmann's solution, in all children over 1 month of age.

- The majority of children over 1 month of age will maintain a normal blood sugar if given nondextrose-containing fluid during surgery. Children at risk of hypoglycemia (if nondextrose-containing fluid is given) may be due to the following reasons:

- On parenteral nutrition
- Received dextrose-containing fluid prior to surgery
- Children of low body weight (<3rd percentile)
- Children undergoing a surgery of >3 hours' duration
- Children having extensive regional anesthesia.

These children at risk should be given dextrose-containing solutions or have their blood glucose monitored during surgery:

- Blood loss during surgery should be replaced initially with 10–20 mL/kg crystalloid or colloid, and then with blood once the hematocrit has fallen to 25%. Children with cyanotic congenital heart disease and neonates may need a higher hematocrit to maintain oxygenation.
- Dehydration without signs of hypovolemia should be corrected slowly.
- Fluid therapy should be monitored by daily electrolyte estimation, use of a fluid input/output chart, and daily weighing, if feasible.

Q. How will you manage the pain postoperatively in patients with CL repair? Can you describe the infraorbital nerve block?

Ans. The infraorbital nerve is 1 cm inferior to the infraorbital ridge and in the midpupillary line (vertical line passing through the supraorbital foramen, pupil, the infraorbital, and the mental foramina). Infraorbital nerve is a branch of the maxillary division of the trigeminal nerve. The infraorbital nerve innervates the upper lip, medial side of the cheek, lateral aspect of nose, and lower eyelid (Fig. 4).

It can be performed in two different ways:

1. *Direct cutaneous injection:* The infraorbital foramen should be palpated, and approximately 1–2 mL of 0.25 or 0.5% bupivacaine is injected near to surround the nerve, but not in the foramen.
2. *Intraoral injection:* During palpation of the foramen with the nondominant hand, the needle is inserted into the superior labial sulcus at the apex of the canine fossa.

Approximately 1–2 mL of 0.25 or 0.5% bupivacaine is injected around the infraorbital foramen. Supplemental systemic analgesics as mentioned earlier.

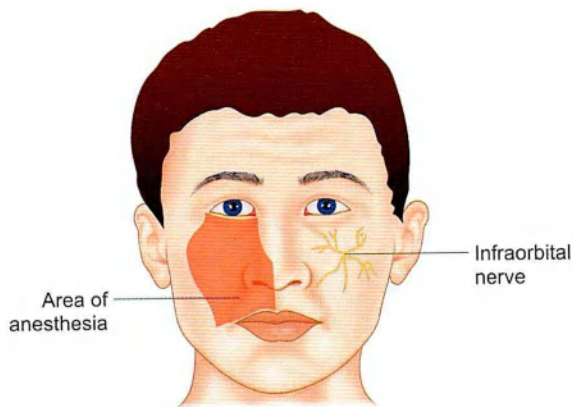


Fig. 4: Infraorbital nerve block.

Q. If a patient with repaired CLP presents for a noncleft surgery (tonsillectomy), what are the anesthetic implications?

Ans. Detailed evaluation should be done as mentioned earlier. Important considerations relevant to this case are mentioned below:

- The child may have received multiple anesthetics before, it is important to go through the notes and retrieve any relevant information with regards to difficult airway, co-existing conditions, any problems faced in the postoperative period.
- It is important to elicit the nature of surgery which was performed, e.g., pharyngoplasty. This operation reduces the size of the space between the soft palate and

pharyngeal wall. This predisposes them to obstructive sleep apnea. One must enquire about snoring.

- Nasotracheal intubation should be avoided in these cases. If a nasotracheal intubation is anticipated, it is safer to do nasopharyngoscopy to evaluate the nasopharynx.

SUGGESTED READING

1. APA Consensus Guideline on Perioperative Fluid Management in Children v 1.1 September 2007 © APAGBI Review Date August 2010.
2. Practice Guidelines for Preoperative Fasting and the Use of Pharmacological Agents to Reduce the Risk of Pulmonary Aspiration: Application to Healthy Patients Undergoing Elective Procedures. A Report by the American Society of Anesthesiologists Task Force on Preoperative Fasting. *Anaesthesiology*. 1999;90(3):896-905.
3. Rawlinson E. Anaesthesia for Cleft Lip and Palate. [online] Available from https://www.anaesthesiak.com/Documents/Cleft_lip_%20&_%20palate_%20s.doc.pdf. [Last accessed February, 2020].
4. Somerville N, Fenlon S. Anaesthesia for cleft lip and palate surgery. *Continuing Education in Anaesthesia Critical Care and Pain*. 2005;5(3):76-9.
5. Steward DJ. Anesthesia for patients with cleft lip and palate. *Seminars in Anesthesia, Perioperative Medicine and Pain*. 2007;26(3):126-32.
6. Tewfik TL, Lacroix Y, Nathalie T. Pierre Robin Syndrome. [online] Available from <http://emedicine.medscape.com/article/844143-overview>. [Last accessed February, 2020].
7. Tolarova MM, Wong GB, Varma S. Mandibulofacial Dysostosis (Treacher Collins Syndrome): Treatment and Medication. [online] Available from <http://emedicine.medscape.com/article/946143-overview>. [Last accessed February, 2020].
8. Tremlett M. Anaesthesia for cleft lip and palate surgery. *Curr Anaesth Crit Care*. 2004;15:309-16.

Geriatric Patient

Amol Trimbakrao Kothekar, Raghu S Thota, Sukhada Savarkar

Medical advances in recent years have increased the life expectancy of the people over the past century. As the population ages, more surgical procedures will be performed on these patients. Although many older adults are quite active and continue to work, they have limited physiological reserve whereas many others have multiple disease processes and are at an increased risk over the general population. This makes it essential to develop a better understanding and increased expertise in caring for the geriatric surgical patient.

Q. Who is a geriatric patient?

Ans. There is no universally acceptable definition of the geriatric patient. The World Health Organization (WHO) defines the elderly or older person as someone who is >65 years of age and a person with age >80 years is called oldest-old. Few authors consider a person aged >60 years as a geriatric patient.

Q. What are the physiological changes due to aging relevant to anesthesia?

Ans. A variable degree of physiological changes of aging affect different organ systems. The changes in individual organ systems are as follows:

- *Cardiovascular and autonomic nervous system:*

- Heart:

- *Myocardium:* There is an increase in myocardial thickening and stiffness with decreased elasticity. This leads to impairment in diastolic relaxation leading to age-related diastolic dysfunction. Diastolic dysfunctions in the elderly can occur without any comorbidities but are more common with coexisting comorbidities such as hypertension, ischemic heart disease, hypertrophic cardiomyopathies, valvular heart disease, and diabetes.
- Elderly patients have elevated filling pressure due to the presence of diastolic dysfunctions. There is decreased early diastolic filling and higher contribution of atrial kick for left ventricular (LV) filling. Elderly patients poorly tolerate a sudden decrease in preload (volume loss) and

arrhythmias like atrial fibrillation (AF). On the other hand, due to elevated filling pressures, elderly patients are at risk of pulmonary edema and poorly tolerate rapid volume expansion.

- *Conductive system:* There is a decrease in the number of conduction fiber density and the number of sinus node cells. It results in decreased beta-adrenergic sensitivity. As age advances, the ability of the sinoatrial (SA) node to increase the heart rate also falls down.
- Elderly patients have a higher incidence of sick sinus syndrome and conduction blocks due to loss of SA node cells and fibrosis of the conduction system. The risk of atrial fibrillation (AF) also increases with age.
- Thickening and calcification of the aortic valve is common in the elderly. The incidence of aortic stenosis also increases with age and is associated with increased risk of adverse cardiovascular and coronary events. These patients poorly tolerate a combination of hypotension and tachycardia.
- *Vasculature:*
 - Vascular stiffness increases with advancing age due to breakdown of elastin and collagen. There is an increase in the diameter and stiffness of large elastic arteries because of which the mean arterial pressure and pulse pressure increase. This increased stiffness in vasculature also causes important secondary changes in the heart. As the resistance in the blood vessels increases,

the velocity of conduction of the pulse waves also increases down the vascular tree. Normally in young adults, reflected waves reach the heart after aortic valve closure. This helps in maintaining pressure in the central aorta and improving coronary blood flow during diastole. With age-related increased vascular stiffness, the pulse wave velocity increases which results in earlier reflection of pulse waves from the periphery. These reflected pulse waves reach the heart during the latter phases of ejection, resulting in an increased workload to the heart. These changes in the afterload of the left ventricle can cause LV wall thickening, hypertrophy, and impairment of diastolic filling.

- There is a decrease in the compliance of the venous system that hinders response to changes in the intravascular volume as experienced during position changes, third space losses, or hemorrhage.
- Autonomic:
 - Aging causes decreased responsiveness to β -receptor stimulation and an increase in the activity of the sympathetic nervous system. The decrease in the β -receptor affinity and altered signal transduction is responsible for this reduced response to β -receptor stimulation. Normally, exercise- or stress-induced sympathetic stimulation causes an increase in the heart rate, venous return, and systolic arterial pressure. The preload reserve is normally unchanged. But in the elderly, due to decreased β -receptor responsiveness, these "high-flow demand" situations are associated with decreased maximal heart rate and decreased peak ejection fraction. Hence, the increased flow demand is met mainly by preload reserve in the elderly, making them prone to heart failure.
 - With advanced age, there is a decrease in the heart's inotropic and chronotropic response to endogenous and exogenous catecholamines.
 - Changes in the α -receptor response are not well established.
 - Overall, these autonomic changes can lead to intraoperative lability and reduced ability to cope with increased metabolic demands during surgery.
 - All these changes in the vascular system are accelerated in the presence of comorbidities such as hypertension, atherosclerosis, diabetes, tobacco abuse, and obesity.
- *Respiratory system:*
 - Structural changes:
 - There is a decline in the function of pharyngeal and laryngeal structures due to loss of muscular pharyngeal support predisposing the elderly to upper airway obstruction.
 - There is an increased incidence of periodic breathing during sleep (obstructive sleep apnea), which makes them more likely to have apnea and airway obstruction in the recovery room.
 - Protective reflexes involved in the regulation of coughing and swallowing are diminished with age. Also, there is loss of ciliary function. Both these factors combined result in repeated aspirations and chronic pulmonary inflammation.
 - The thorax changes shape with age. Kyphosis of the thoracic spine is the first change, beginning around the age of 50 years in females and a decade later in males, and is due principally to osteoporotic vertebral collapse. This leads to an increase in the anteroposterior diameter of the chest at the expense of the lateral diameter. These changes lead to a typical barrel chest appearance. There is also flattening of the diaphragm. There is significant loss of muscle mass due to aging which adds to the functional impairment of diaphragm. Costochondral joints get calcified with aging. The compliance of the chest wall is reduced due to these changes. This leads to increased work of breathing.
 - Lung compliance increases due to loss of elastin with age and altered production of the surfactant. Since airways lose elastic support, they become more collapsible and larger numbers of airways close during expiration, particularly in the lung bases. Closing capacity, the lung volume at which there is collapse of the small airways increases with age. By 60–70 years of age, the closing capacity exceeds the functional residual capacity (FRC) causing reduction in expiratory flows at the end of expiration.
 - There is loss of elastic elements in the lung parenchyma, enlargement of respiratory bronchioles and alveolar ducts, and increase in the size of the interalveolar pores of Kohn, leading to progressive loss of the alveolar surface area. These changes result in increased dead space, decreased diffusing capacity, and increased closing capacity.
 - *Lung volumes and capacities:* Aging produces an increase in the residual capacity by 5–10% per

decade. There is a decrease in the vital capacity, but the total lung capacity remains relatively unchanged. The reduction in motor power of the accessory muscles of breathing as well as the decreased expansion of the chest wall causes the dynamic lung volumes and capacities to decrease progressively with age. The forced expiratory volume in 1 second reduces with age by 6–8% per decade.

- *Gas exchange:* There is impairment of gas exchange with aging due to a decrease in the diffusion capacity, increased dead space, and increased closing capacity.
- *Alveolar-arterial gradient:* Aging is associated with an increased alveolar-arterial oxygen gradient. As age progresses, the relationship between FRC and closing capacity changes. Normally, the closing capacity is lower than FRC in the young. As the age progresses, the closing capacity equals FRC initially in the supine position and later (at 66 years of age) in the upright position. When the closing capacity becomes more than FRC, shunt occurs which ultimately leads to decreased arterial oxygenation. This change between FRC and closing capacity causes ventilation-perfusion mismatch. It is the most important reason for an increase in the alveolar-arterial oxygen gradient. Mean PaO_2 declines from 95 ± 2 mm Hg at the age of 20 years to 73 ± 5 at the age of 75 years. This decline in arterial oxygen tension is modest, approximately 0.4 mm Hg/year. After the age of 75 years, however, PaO_2 stays relatively constant at approximately 73 mm Hg.
- *Hypoxic pulmonary vasoconstrictor (HPV) reflex and pulmonary vasculature:* There is a decrease in the cross-sectional area of the pulmonary capillary bed as the age progresses. Increase in the pulmonary vascular resistance and pulmonary arterial pressure associated with aging can be because of that. This increasingly rigid pulmonary vasculature blunts the HPV reflex. Thus, the ability of the aged lung to respond to an altered ventilation-perfusion ratio is compromised. This effect is more highlighted in one-lung anesthesia.
- *Respiratory drive:* Due to decreased central nervous system (CNS) activity, there is impairment of ventilatory responses to hypoxia, hypercapnia, and stress. Elderly patients have increased sensitivity to respiratory depressant effects, opioids, benzodiazepines, and volatile anesthetics.

Overall, the age-related changes of the respiratory system essentially consist of a combination of restrictive and obstructive lung diseases resulting in impaired gas exchange and decreased arterial oxygenation as age progresses.

- Central nervous system:
 - Brain:
 - Structural changes:
 - ♦ Aging causes cerebral atrophy which is selective in nature. The volume of gray and white matter decreases. In the gray matter, neuronal shrinkage contributes more to the volume loss than the actual loss of neurons.
 - ♦ There can be white matter loss of around 15% which results in dilated ventricles and atrophy of gyri.
 - ♦ Neuronal shrinkage is seen in the subcortical white matter and the hippocampus. This can be aggravated in the presence of other comorbidities such as hypertension.
 - Biochemical changes:
 - ♦ Nonhuman studies suggest that aging causes regional depletion in the neurotransmitter levels of dopamine, acetylcholine, norepinephrine, and serotonin, but the levels of glutamate are unchanged. The association of serotonergic, cholinergic, and dopaminergic systems, respectively, with mood, memory, and motor function may partially account for depression, loss of memory, and motor dysfunction in the elderly.
 - ♦ The cerebral blood flow and cerebral oxygen consumption (CMRO_2) decreases in proportion to the decrease in brain mass. However, the cerebral autoregulation is well maintained in geriatric patients without prior neurological disease.
 - Sleep: Increased latency to sleep onset is often present as well as increased awakenings and periods of wakefulness during the night.
 - Memory: Memory decline occurs in >40% of adults who are older than 60 years of age.
 - Age-related CNS diseases: Age-related diseases such as cerebral arteriosclerosis and Alzheimer's and Parkinson's disease are all more common with advancing age.
 - Perception: Aging produces a generalized increase in the thresholds for virtually all forms of perception, including vision, hearing, touch, sense of joint position and smell as well as peripheral pain and temperature responses. Elderly are likely to under-report acute postoperative pain. Nevertheless, it should not be undertreated as it may have adverse consequences similar to the younger

population. There might be reduction in the total dose requirement of analgesics. Nerve blocks and neuraxial blocks should be used whenever possible to reduce the requirement of opioids as higher doses of opioids are associated with respiratory impairment, ileus, and suboptimal mobilization.

- *Functional brain reserve:* There is a decrease in the functional brain reserve with aging. Its exact mechanism is not known. This decrease in reserve can predispose elderly patients to increased sensitivity for anesthetic agents, increased prevalence of postoperative delirium and cognitive dysfunction, delayed discharge, and functional recovery after surgery.
- *Thermoregulation:* Elderly patients are particularly at risk for hypothermia in an operating room environment due to impaired vasoconstriction and decreased metabolism. Prevention of hypothermia is of utmost importance in these patients. Hypothermia increases the risk of delayed recovery due to reduced drug metabolism and can perpetuate into coagulopathy with increased blood loss leading to further hypothermia. There is increased risk of myocardial ischemia and surgical wound infection with hypothermia. Hypothermia also leads to shivering that may not be tolerated by patients with borderline cardiac or pulmonary reserve due to increased metabolic demands.
- *Neuraxial system:*
 - Aging causes reduction in the area of the epidural space and increase in the permeability of the dura. The volume of cerebrospinal fluid (CSF) reduces in elderly patients. Aging also causes reduction in the diameter and number of myelinated fibers in the dorsal and ventral nerve roots.
 - Neuraxial block procedures can be more difficult in the elderly due to various reasons. There can be difficulty in positioning due to age-related reduction in joint mobility; needle insertion, especially in midline approach, can be challenging due to sclerotic changes; and calcification of ligamentum flavum. Sensitivity to local anesthetics can be increased due to increased permeability of dura, reduction in the number of neurons within the spinal cord, and deterioration of myelin sheaths and connective tissue barriers. As discussed previously, these patients poorly tolerate a sudden decrease in preload and are at greater risk of hypotension with neuraxial blocks. The elderly patients are more prone to hypothermia with neuraxial blocks.
- *Peripheral nervous system and neuromuscular function:*
 - The inter-Schwann cell distance in peripheral nerves is decreased in the elderly. The conduction velocity of peripheral nerves is also reduced due to aging.
 - Motor neuron degeneration produces disseminated neurogenic atrophy responsible for age-related changes at the neuromuscular junction. Thickening of the postjunctional membrane and its spread beyond the usual end-plate areas because of age-related deficiency of cyclic guanosine monophosphate (GMP) generate atypical "extrajunctional" cholinergic receptors. The increase in the number of cholinergic receptors at the end plate and surrounding areas might compensate for the age-related decline in the number and density of motor end-plate units. Therefore, despite loss of skeletal muscle mass, the dose requirements for competitive neuromuscular blocking drugs are not reduced.
- *Renal system:*
 - There is a progressive reduction of renal mass with aging (30% by 80 years of age) due to glomerulosclerosis, paralleled by thickening of the vascular intima, and chronic infiltration by inflammatory cells. This results in a decline in renal plasma flow and glomerular filtration rate (GFR). There is about 10% decrease in the renal blood flow per decade.
 - Microscopically, there is a reduction in the number of functional glomeruli.
 - In spite of reduction in renal function, serum creatinine levels remain relatively normal due to a concomitant decrease in muscle mass. Hence, the serum creatinine level is a poor indicator of GFRs in the geriatric population.
 - From the age of 40 years, there is a drop of roughly about 1 mL/min/m² in GFR every year. The Cockcroft Gout formula can be used to estimate GFR. $[GFR = (140 - \text{age}) \times \text{Wt}/72 \times \text{serum creatinine}]$. Thus, for a 68-year-old person weighing 60 kg with serum creatinine 1.2, the estimated GFR is 50 mL.
 - The reduced blood flow and loss of the total available mass of nephron units in the elderly also delay the drug clearance and prolong the clinical effects of drugs, which require final elimination by renal pathways.

- Renal vascularity is reduced and the cardiac output is redistributed, predisposing the geriatric population to renal ischemia in the perianesthetic period.
- Variation in the decline of renal function with age is very common. This is secondary to the nephrotoxic effects of comorbidities such as diabetes and hypertension and drugs, especially nonsteroidal anti-inflammatory drugs (NSAIDs) and angiotensin-converting-enzyme (ACE) inhibitors. Renal function should be routinely assessed as it affects the pharmacokinetics and pharmacodynamics of anesthetic drugs.
- Elderly are more prone to urinary retention which can be significant enough to cause postoperative agitation. Urethral catheterization can be difficult or sometimes traumatic in the elderly due to higher prevalence of benign prostatic hyperplasia (BPH).
- Elderly are vulnerable to sudden loss or excess of both sodium and free water. Major fluid and electrolyte shift or rapid infusion of nonisotonic fluids should be avoided if possible. Frequent electrolyte monitoring may add a layer of safety.
- Geriatric patients have reduced abilities to concentrate urine or conserve sodium. Elderly patients tend to lose sodium even in the presence of inadequate salt intake. Their thirst mechanism is also reduced. Hence, they are at increased risk of dehydration and sodium depletion. Aging also reduces the ability of the elderly to deal with increased salt load. This may result in increased sodium retention and extracellular volume expansion in the perioperative period.
- Loss of lean body mass also reduces the total body stores of exchangeable potassium and predisposes to iatrogenic hypokalemia. The elderly tolerate hypokalemia poorly owing to age-related myocardial and conduction tissue dysfunctions. Thus, the fluid and electrolyte status should be carefully monitored in the elderly patient.
- *Gastrointestinal system:*
 - The gastrointestinal system changes of the geriatric patient maybe characterized by gastric acidity, decreased colon motility and anal function, constipation, fecal impaction, fecal incontinence, osteoporosis, or vitamin B₁₂ deficiency.
- *Hepatic system:*
 - Liver tissue decreases approximately 20–40% as the age progresses, which leads to decreased hepatic function. There is a decline in the hepatic blood flow of around 10% per decade.
 - There is a lack of correlation between structural and functional data concerning the aging liver, as a decline in organ volume does not necessarily reflect impaired metabolic function. The intrinsic capacity of the liver to metabolize drugs is reduced variably.
- *Drug metabolism through phase-1 pathways such as oxidation, reduction, and hydrolysis through the cytochrome P450 system can be affected due to aging. Metabolism of drugs such as ketamine, opioids, and lignocaine that have high hepatic extraction can be slower in the elderly due to reduced hepatic blood flow. Drugs with hepatic metabolism in the form of acetylation or conjugation are least affected.*
- *Body compartments and musculoskeletal system:*
 - There is loss of skeletal muscle (decrease in lean body mass) and decrease in total body water mainly due to a decrease in intracellular water.
 - Plasma volume, red cell mass, and extracellular fluid volumes are well maintained in nonhypertensive elderly individuals with adequate levels of physical activity.
 - The percentage of body fat increases with aging. This acts as a reservoir for the deposition of lipid-soluble anesthetic drugs, increasing the elimination time and prolonged recovery time.
 - Age-related decreased muscle mass, bone density, and lubrication of the joints cause stiffness of the joints, osteoporosis, frequent fractures of the hip, and functional impairment of bones and joints. Extra care and attention must be given to positioning and padding the patient because of fragile skin, decreased subcutaneous fat, and poor skin turgor. In addition, age-related changes in the musculoskeletal system and limitations often imposed by chronic pain in the elderly make patient positioning an extremely important aspect of care.
 - Impaired rehabilitation and immobility increase chances of thromboembolism and pressure necrosis.
- *Endocrine system and metabolism:*
 - Overall, there is a decreased activity of the endocrine system, causing impaired glucose homeostasis; decreased thyroxine clearance or production; decreased production of renin, aldosterone, and testosterone; decreased vitamin D absorption and activation; and increased plasma concentration of the antidiuretic hormone.
 - Basal resting metabolic activity decreases in direct proportion to the age-related decrease in lean tissue mass.
 - Thermoregulation is affected due to autonomic impairment, as well as changes in the skin and blood vessels, leading to increased heat loss and

reduced heat tolerance. As a consequence, the elderly patients are vulnerable to heat stroke and hypothermia. Also, the time required for spontaneous rewarming postoperatively appears to increase in direct proportion to a patient's age. Therefore, maintaining normothermia is of special concern in the geriatric patient.

- There is glucose intolerance secondary to a progressive impairment of insulin function or subtle antagonism of its effect on the target tissues with aging.
- The age-related progressive impairment of the ability to handle an intravenous glucose challenge may also be in large part due to a decrease in lean body mass tissue as this tissue normally provides storage for carbohydrates. The renal threshold for glucose is notoriously unpredictable in the elderly, and urine testing is not a reliable sign of the presence or absence of hypo/hyperglycemia.
- **Hematological and immune system:**
 - There is reduction in bone marrow production and spleen size, which reduces the hematopoietic response to imposed anemia possibly related to erythropoietin resistance and stem-cell aging.
 - **Immunosenescence:** The multifactorial deterioration of the immune system leading to reduction of the capacity to counter infection and wound healing is known as immunosenescence. With age, there is depression of the selectivity and the effectiveness of the immune response making the elderly susceptible to life-threatening infection. There is anatomic involution of the thymus gland and altered function of T lymphocytes. Elderly patients are known to have atypical presentation of diseases. Typical response to infections such as fever and rise in white blood cell (WBC) count might be less pronounced or maybe absent in elderly patients.

Q. Describe the alterations in the pharmacokinetics (PK) and pharmacodynamics (PD) of various anesthetic drugs due to aging.

Ans. There are multiple physiological changes that alter the ability of the elderly to handle pharmacologic agents (**Table 1**). The apparent age-related increase in potency in the anesthetic drugs maybe due to a pharmacodynamic phenomenon (e.g., change in the beta-receptor sensitivity resulting in a reduced chronotropic and ionotropic response to beta-adrenergic drugs, thus requiring increased doses) or it may just be a result of the pharmacokinetic changes (continual loss of neuronal substance accompanied by a parallel reduction in the CMRO₂) necessitating an alteration in drug doses.

TABLE 1: Alterations in the pharmacokinetics and pharmacodynamics in the elderly.

Pharmacokinetic/ pharmacodynamic alteration	Effect	Drugs
Slower redistribution due to decrease in muscle mass and lower blood volume	Higher peak effect of bolus drug for longer time	Thiopentone propofol
Slow plasma-brain equilibrium possibly due to delayed transfer across blood-brain barrier and slower circulation	Delayed peak effect of bolus drug	Remifentanyl
Decreased drug metabolism due to decreased renal and hepatic clearance Increased Vd _{ss} due to increased body fat	Prolonged and residual effects of large, repeated doses or prolonged infusions Increased context-sensitive half-time	More common with fat-soluble drugs
Possible increased sensitivity of target organ (brain)	Lesser requirement of drugs	Opioids, propofol, and midazolam

(PK/PD: pharmacokinetics and pharmacodynamics; Vd_{ss}: volume of distribution at a steady state).

Other factors that affect the pharmacokinetics and pharmacodynamics are as follows:

- **Changes in the cardiovascular system:** Elderly individuals are more susceptible to the hypotensive effects of anesthetic drugs. There is delayed induction of anesthesia following the administration of intravenous anesthetic agents due to reduction in cardiac output. In contrast, inhalational inductions are faster due to the rapid rise of the alveolar partial pressure, which would in turn help in achieving higher brain partial pressures of inhalational agents.
Hypotension after induction maybe compounded if the patient is on beta-blockers, calcium entry blockers, nitrates, or ACE inhibitors:
- **Renal and hepatic effects of aging:** The renal clearance is reduced in the elderly subjects even though they maintain normal serum creatinine values. Thus, the elderly will have a delay in the offset of drugs and their metabolites excreted by the kidney, e.g., pancuronium and pethidine.

Most of the intravenous drugs used in the practice of anesthesia are metabolized in the liver. Aging is associated with a decrease in the liver volume, liver blood flow, and the intrinsic hepatic capacity, all leading to a reduced total drug clearance and a higher steady-state plasma concentration in the elderly.

Protein binding: Albumin and alpha-1-acid glycoprotein are the primary sites of plasma protein binding. There is a reduction in the plasma albumin concentration even in the well-nourished healthy elderly individual. This change

is exaggerated in the poorly nourished and those with advanced illness. As a result, a greater quantity of free drug is available for diffusion to the site of action. Therefore, acid drugs that bound to albumin, such as pethidine and diazepam, have reduced dose requirements in the elderly. The increased alpha-1-acid glycoprotein in the elderly reduces the free fraction of lignocaine and retards clearance in the elderly. Therefore, these changes in protein binding have an impact on the free fraction, clearance, and the volume of distribution of drugs.

Change in the pharmacology of specific drugs:

- **Thiopentone sodium:**
Decrease in the lean body mass → Reduction in the volume of distribution → High plasma concentrations → Reduced dose required to achieve anesthesia:
 - Thiopental induction doses should be about 85% of the younger counterparts.
 - Prolonged infusion of thiopentone will lead to accumulation of drug.
- **Propofol:**
 - Elderly brain is more sensitive to propofol.
 - Smaller central compartment and a reduced volume of distribution.
 - Reduced clearance:
 - Induction dose reduced (around 1.7 mg/kg)
 - Maintenance dose reduced by 30–50%.
- **Benzodiazepines:**
 - Midazolam, lorazepam, and diazepam have comparable protein binding and volume of distribution.
 - However, the high clearance rate of midazolam makes it an attractive alternative.
 - A reduction of about 75% in the midazolam dose has been suggested due to increased sensitivity of the brain and a reduced clearance.
- **Opioids:**
 - Twice as potent in the elderly subjects
 - 50% reduction in doses
 - Shorter acting opioids, i.e. fentanyl, alfentanil, sufentanil, and remifentanil, are better choices.
- **Muscle relaxants:** The pharmacodynamics of muscle relaxant agents does not change much with advancing age; however, the duration of action maybe prolonged in the elderly patients for the drugs whose clearance depends on liver metabolism or renal excretion. The clearance of atracurium or cisatracurium is unchanged in the elderly and does not require dose modifications. The dose of neostigmine for reversal of neuromuscular blockade is unchanged; however, elderly patients are more prone to the adverse cardiac effect of this drug:
- **Inhalational agents:** Age-related changes in the cerebral metabolism reduce the minimum alveolar

concentration (MAC) of the inhaled anesthetics. The decreased cardiac output in the elderly as mentioned earlier along with a decrease in the MAC values results in rapid induction rates.

Recovery from inhalational agents is prolonged and has a pharmacokinetic basis. The decrease in the lean body mass and an increase in the body fat increase the volume of distribution, especially for fat-soluble agents. In addition, decreased hepatic function together with a decreased pulmonary gas exchange may decrease the anesthetic clearance with age:

- **Other drugs:** Due to reduced vagal tone, higher doses of atropine maybe required to achieve effect similar to a young patient. Due to higher risk of central cholinergic syndrome, glycopyrrolate, which does not cross the blood-brain barrier, maybe preferred over atropine.

Adverse drug interaction: Balanced anesthesia requires the administration of various drugs. The possibility of adverse drug reaction increases with polypharmacy used for balanced anesthesia in the elderly due to the decreased physiological reserve as well as the concomitant drug therapy that they commonly take. Hence, an anesthesiologist needs to be aware of possible drug interactions to avoid serious adverse reactions.

Q. Describe the perioperative risks involved with respect to old age.

Ans. Perioperative risk is a combined effect of surgical procedure and patient factors.

Risk related to the surgery: Emergency surgery is likely to pose more risk than elective surgery in the elderly as elective surgery allows preoperative optimization of modifiable risk factors. Also the nature of surgery, invasiveness, and extent of surgical resection would play an important role in perioperative outcome.

Risk related to patient: Elderly patients are more prone to postoperative morbidity and mortality because of limited physiological reserve, associated medical comorbidities, frailty, and postoperative cognitive dysfunction (POCD).

Q. What factors influence regional anesthesia techniques in the old age?

Ans.

- **For neuraxial blocks:**
 - Size of the epidural space is reduced.
 - Permeability of the dura is increased.
 - Volume of the CSF is decreased.
 - There is a decrease in the size and number of myelinated fibers in the dorsal and ventral nerve roots.

- Narrowing of intervertebral spaces and growth of osteophyte decrease transforaminal escape resulting in more cephalad spread of local anesthetic agent in the epidural space.
- Due to above-mentioned reasons, the onset of neuraxial anesthesia is more rapid and the level of spread is more extensive, especially with the hyperbaric solutions.
- Higher plasma concentrations of LAs after epidural anesthesia have been observed in the elderly people. This may reflect prolonged elimination half-life, resulting in reduced plasma clearance. Because of reduced clearance, toxic plasma concentrations may result from lower doses of LA drugs in elderly patients.
- *For local anesthesia/peripheral nerve blocks:*
 - There is a decrease in the conduction velocity of the peripheral nerves owing to a decrease in the inter-Schwann cell distance.
 - There is a decrease in the number of axons in the peripheral nerve.
 - All these factors suggest a generalized reduction in the requirements for LA drugs in a variety of regional techniques.

Q. How will you evaluate and assess a geriatric patient preoperatively?

Ans. During the preanesthesia checkout, the following points are important (**Table 2**):

- *To assess the functional status of these patients:* Baseline preoperative functional status should be assessed by taking careful history, assessment of activities of daily living, clinical examination, and laboratory tests whenever required (6-minute walk test, cardiopulmonary exercise testing, etc.).
- *To identify commonly associated comorbid conditions and optimize the patient accordingly before surgery:* Multiple diseases are the rule rather than exception in elderly patients. A variety of chronic disorders, such as hypertension, diabetes mellitus, atherosclerosis, myocardial infarction, cerebrovascular accidents (CVA), pulmonary diseases, osteoporosis, osteoarthritis, anemia, Parkinsonism, dementia, and even malignancy, are observed with increasing frequency in this patient group. Assessment should provide the information needed to determine the severity of pre-existing disease and the adequacy of a remaining organ system reserve. This would help in risk stratification as well as optimization of a patient before surgery.

TABLE 2: Preoperative assessment of geriatric patient.

Domain	Features to be assessed	Appropriate assessment tools
Cognitive functions	• Mental capacity • Decision-making capacity • Communication • Risk factors for postoperative delirium	• Mini-Cog based on clock drawing and three-word recall • Ask "Have you (or your carer) noticed a change in your memory?" • Abbreviated mental test score • Vision, hearing, speech • CAGE questionnaire for alcoholism • American College of Surgeons National Surgical Quality Improvement Program (ACS NSQIP) preoperative assessment
Functional capacity	• Gait and balance • Mobility • Frailty	• 6-m walk • Walks unaided/with stick/with frame/does not walk housebound? (yes/no)
Use of functional aids	• Visual • Hearing • Mobility • Dentures	• Glasses • Hearing aids • Walking stick, frame, wheelchair
Risk score	• Pathology specific • Frailty	• Nottingham Hip Fracture Score • NSQIP preoperative assessment

- *Frailty:* Frailty is found in around 20–30% of the elderly population >75 years of age, and it increases with advancing age. It has been linked to increased risk of deaths, falls, and institutionalization. The NCEPOD (National Confidential Enquiry into Patient Outcome and Death) 2010 report gives an operational definition of frailty as per the Canadian Veterans Heart Study Definition of frailty, which states that frailty exists when the patient displays any three of the following conditions:

- Unintentional weight loss (at least 4 kg in the last year)
- Self-reported exhaustion
- Weak grip strength
- Slow walking speed
- Low physical activity.

There are no specific, validated, preoperative tests for frailty. The most common method of assessing frailty is by history and examination.

Once frailty has been identified in a patient, its assessment and management require multidisciplinary team effort which should include

surgeons, anesthesiologists, nursing, rehabilitation, nutritionists, and physicians.

- *Review of medication:* Elderly patients are likely to be on multiple medications due to associated comorbidities. Careful review is necessary to avoid interactions and side effects.
- A preoperative assessment should also include *mental preparation* of the patient and relatives before surgery, explaining the need for postoperative ventilation and intensive care unit (ICU) stay in high-risk surgeries, and likelihood of the patient returning to baseline functional status.
- Presence of artificial dentures, hearing aids, etc., should be noted. Dependence on others for daily activities should be noted.
- History of fall is an important predictor of perioperative complication.

Readers are referred to the American College of Surgeons National Surgical Quality Improvement Program and the American Geriatrics Society checklist 2012 for the Optimal Preoperative Assessment of the Geriatric Surgical Patient. This checklist should be followed over and above the routine assessment. Few important points in the checklist include:

- Cognitive ability and capacity to understand
- Depression, delirium, substance abuse/dependence
- Functional status and history of falls, frailty score
- Nutritional status.

Q. What is the choice of anesthesia?

Ans. Type of anesthesia: The choice of anesthesia—regional or general—appears to be of less importance than how meticulously and carefully it is administered with regard to the patient's functional status. Observational studies and meta-analyses do not reliably show any significant difference in the outcome between regional anesthesia and general anesthesia (GA), but this maybe because the outcomes measured are not specifically related to anesthesia (e.g., 30-day mortality and length of stay) or because regional anesthesia is seldom administered without sedation. The choice of technique, therefore, should be made according to the individual patient.

Regional anesthesia is an attractive option as it may reduce intraoperative and postoperative opioids' consumption, pulmonary complications (atelectasis, pneumonia, and prolonged mechanical ventilation), and stress (tachycardia and hypertension). Regional anesthesia may reduce the incidence of POCD; however, evidence for its support is lacking. On the other hand, if regional anesthesia, especially neuraxial blockade, is not performed meticulously and carefully, the resultant hypotension and hypothermia can be detrimental.

A multimodal nonopioid-based regimen (paracetamol, NSAIDs, and regional or neuraxial blocks whenever appropriate) for postoperative analgesia reduces reliance on opioids and its adverse effects.

Q. Is the Surgical Safety Checklist in Geriatric Patients different from the usual check list?

Ans. The WHO has amended the Surgical Safety Checklist for all patients aged >75 years. Few important points in the checklist include the following:

Before induction:

- Does the patient have dentures?
- What is the patient's core temperature?
- Does the patient have any preoperative pressure sores?

Before incision:

- Have possible areas of pressure damage been padded?
- What is the patient's estimated GFR (eGFR)?

Before the patient leaves the operation theater:

- What is the patient's core temperature?
- What is the patient's hemoglobin concentration?
- Has a postoperative fluid plan been prescribed?

Q. Describe important issues in intraoperative period in the old age.

Ans. Induction agents should be given slowly and titrated, more frequently with the measurement of blood pressure keeping in mind the reduced requirements of anesthetic drugs and increased risk of hypotension. Anesthesia depth monitoring with bispectral index (BIS) or end-tidal gases is highly desirable. Short-acting drugs should be preferred to long- and intermediate-acting drugs. Neuromuscular monitoring is also highly desirable. It is critical to maintain normotension, normoxia, and normothermia intraoperatively.

In all elderly patients, electrocardiogram (ECG), oxygen saturation, temperature, and blood pressure must be monitored. Urine output should be monitored for major surgeries. Direct intra-arterial pressure and advanced hemodynamic monitoring can be instituted early in patients at high risk or those who have developed hemodynamic instability. Warm peripheries (toes), coupled with an hourly urine output >0.5 mL/kg/h, usually imply a satisfactory hemodynamic status.

Hypotension is a common problem. Patients are often hypovolemic from the preoperative period itself. They maybe hypovolemic due to decreased thirst and inadequate fluid intake, prolonged periods of fasting, and concomitant diuretic therapy. This may get exacerbated perioperatively due to reduction in autonomic activity and insufficient compensation. The volume of fluid that maybe necessary to support the circulation during the anesthetic may prove excessive once the anesthetic is eliminated and

the sympathetic tone returns, exposing the patient to a risk of volume overload and pulmonary edema. It is safer to administer volume in small intermittent boluses, watching the response of blood pressure, urine output capillary refill, arterial lactate, etc.

There is a narrow margin of safety for fluid management. Intraoperative hypotension is primarily due to vasodilatation unless there is major blood loss or fluid shift. Fluids, vasopressors, or both in combination are used to treat intraoperative hypotension. There is no fixed formula to decide one over another. As discussed earlier, large volumes of fluids given intraoperatively to compensate for vasodilation and to maintain cardiac output and blood pressure may translate into postoperative hypervolemia which increases the risk of pulmonary edema.

A combination of low concentrations of volatile anesthetics, lower BIS score indicating a deep hypnotic level, and low blood pressure (known as triple low) is associated with longer hospital stay and increased 1-month mortality.

Normoxia should be maintained with optimum oxygen concentration. It may be prudent to avoid higher oxygen concentrations. Low tidal volume with positive end-expiratory pressure (PEEP) should be used particularly for intra-abdominal surgeries.

Except for short procedures, temperature should be monitored and hypothermia should be aggressively prevented and treated if necessary.

Q. Describe important issues in the postoperative period in the old age.

Ans. Dexmedetomidine may be preferred to benzodiazepines for postoperative sedation for ventilated patients.

All elderly patients after completion of surgery should be transferred to a recovery room, a high-dependency unit, or an ICU depending on their condition. Just like other patients, the details of the preoperative status, the anesthetic, drugs, doses, and timing of the drug administered during surgery, details of monitoring, other procedures performed, and complications must be noted.

Oxygenation: In elderly patients, the increase in cardiac output and ventilation to satisfy oxygen demand does not occur readily. Diffusion hypoxia may be more serious and prolonged in the elderly after GA, and hence all patients should receive supplemental oxygen.

Postoperative analgesia: Poorly controlled pain in the postoperative period can lead to slow recovery and life-threatening complications. Also, one of the risk factors suggested for POCD is poor pain control. Hence, adequate analgesia in the postoperative period is a mandate.

Nonsteroidal anti-inflammatory drugs (NSAIDs) and paracetamol may be used by intravenous, intramuscular, oral, or rectal route. NSAIDs should be used with care in elderly patients given their cardiovascular, gastrointestinal, and renal adverse effects, and patients should be monitored closely. It is pertinent to mention that the serum creatinine alone poorly reflects renal function in the elderly due to decrease in muscle mass, and the eGFR test may be a better indicator of renal function in these patients. NSAIDs should be avoided in patients with pre-existing renal dysfunction and in those who have suffered hemodynamic instability or major blood loss.

Peripheral blocks when feasible should be used. Intra-articular injection of LAs or narcotics can provide effective analgesia in joint surgeries. Epidural analgesia should be used for surgeries involving thorax, abdomen, pelvis, and lower extremities.

Hemodynamics: The heart rate may not be a reliable index of hypovolemia in the elderly patient. Due to reduced numbers of adrenergic receptors, decreased efficacy of baroreceptor reflexes, and the administration of concomitant beta-blockade and hypothermia, hypovolemia may exist without tachycardia and may manifest with profound hypotension. Patients may have atypical signs of impending hemodynamic instability such as nausea, mental obtundation, and tachypnea.

In patients with major third space loss, by the second postoperative day, fluid starts mobilizing from the third space to the central compartment risking the patients for fluid overload and pulmonary edema. Reduction of intravenous (IV) infusion rate and judicious use of diuretics should be considered on a case-to-case basis based on the clinical signs of fluid overload.

Administration of hypotonic fluids [5% dextrose, 0.33% normal saline (NS), etc.] may result in hyponatremia and low serum osmolality (<235 osmol/L) with resultant risk of cerebral edema.

Arrhythmias are common occurrence in the elderly patients. In patients without preexisting cardiac disease, they may represent disturbances due to hyperventilation (most common cause is pain), hypokalemia, hypomagnesemia, hypocalcemia, hypoxia, or hypercarbia. These metabolic abnormalities can be easily diagnosed and treated. Arrhythmias may indicate myocardial ischemia [especially ventricular premature contractions (VPCs) >5/min, bigeminy, ventricular tachycardia, and various degrees of heart block other than first degree]. AF is not uncommon and should be treated if the ventricular response is rapid or there is hemodynamic deterioration. AF with low blood pressure may require electrical cardioversion. When the blood pressure is well-maintained, calcium-channel

blockers such as diltiazem or beta-blockers can effectively reduce the ventricular response. Minor arrhythmias may be significant in elderly patients with ischemic or valvular disease such as aortic stenosis. Hemodynamic implications include loss of atrial kick in AF, bradycardia worsening a fixed low cardiac output, tachycardia decreasing ventricular filling and increasing oxygen consumption, and VPCs resulting in less effective contraction.

Hypothermia: Hypothermia manifests as altered mental status, delayed recovery from anesthesia, sluggish deep tendon reflexes, and a slow or shallow respiratory pattern. Cardiac rhythm disturbances include supraventricular tachycardia (SVT), AF, atrial and ventricular ectopics, various degrees of heart block and conduction disorders, and suppression of SA node, associated with hypotension and a falling cardiac output. Hypothermia also leads to metabolic disturbances, reduces liver and renal perfusion, induces coagulopathy, and also makes nondepolarizing muscle relaxants more difficult to reverse with anticholinesterase drugs.

Mild hypothermia is best corrected with gradual spontaneous rewarming with a blanket in a comfortably warm room, but severe hypothermia would need active warming methods, such as use of warm IV fluids and surface warming in the postoperative room along with continuous core temperature monitoring.

Postoperative CVA/transient ischemic attack (TIA): Strokes may present as mono/hemi/paraparesis/plegia. Patients may be obtunded or confused. Any mental change persisting or occurring beyond the normal expected period of anesthetic recovery should be viewed suspiciously and needs a thorough clinical examination. Radiological imaging in the form of computed tomography (CT) scan or magnetic resonance imaging (MRI) should be performed, and if there is no evidence of intracerebral hemorrhage, aspirin or heparin may be considered.

Sleep-disordered breathing: The reason for the increased episodes of disordered breathing during sleep with age is unclear. As high as two-third of the elderly have frequent episodes of desaturation and apnea during sleep with diminished response for both hypercapnia and hypoxia. Patients receiving narcotics and sedatives should be closely monitored by pulse oximetry.

Renal system: Elderly patients are at risk for renal failure in the postoperative period following major surgery. The reasons are multifactorial and include prerenal causes such as hypovolemia and cardiac dysfunction. Drugs are important causes of renal dysfunction, and aminoglycosides, ACE inhibitors, NSAIDs, and radiocontrast media are

frequently associated with renal failure. Renal failure may be one of the major manifestations of sepsis.

Miscellaneous: Care must also be given to make the patient as comfortable as possible and to provide him/her with eyeglasses, hearing aids, prosthesis, etc. Bowel and bladder functions must be monitored. Elderly patients may not pass urine even after a minor operation performed under a neuraxial block.

A higher incidence of BPH should be kept in mind.

Deep vein thrombosis (DVT): Elderly patients are more prone to DVT and pulmonary embolism (PE). Advanced age; obesity; prior thromboembolism; malignancy; immobility; pelvic, hip, and orthopedic surgery; central lines; and congenital thrombophilic disorders are other important risk factors. Although more commonly it occurs in the postoperative period, sometimes it can complicate the preoperative or intraoperative period. DVT prophylaxis includes early mobilization, graduated compression stockings (antiembolic stockings), chemical prophylaxis, and pneumatic compression devices. The choice and timing (preoperative, intraoperative, and postoperative) of DVT prophylaxis depend on a patient's mobility, clinical condition, baseline risk factors, and nature of surgery.

Q. What is delirium and postoperative cognitive dysfunction (POCD)?

Ans. Delirium is acute fluctuating cognitive disruption, characterized by confusion with altered attention and awareness which cannot be attributed to pre-existing or developing dementia.

The diagnostic criteria for delirium defined by the American Psychiatric Association's fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) is listed below:

- A. Disturbance in attention (i.e., reduced ability to direct, focus, sustain, and shift attention) and awareness (reduced orientation to the environment).
- B. The disturbance develops over a short period of time (usually hours to a few days), represents an acute change from baseline attention and awareness, and tends to fluctuate in severity during the course of a day.
- C. An additional disturbance in cognition (e.g., memory deficit, disorientation, language, visuospatial ability, or perception).
- D. The disturbances in Criteria A and C are not better explained by a pre-existing, established, or evolving neurocognitive disorder and *do not occur in the context of a severely reduced level of arousal such as coma*.
- E. There is evidence from the history, physical examination, or laboratory findings that the disturbance is a direct physiological consequence of another medical

condition, substance intoxication or withdrawal (i.e., due to a drug of abuse or to a medication), or exposure to a toxin or is due to multiple etiologies.

The screening tools for the detection of delirium in ICU include:

- Confusion Assessment Method-ICU (CAM-ICU) (widely used and validated):
 - Acute onset or fluctuating course
 - Inattention
 - Disorganized thinking
 - Altered level of consciousness.
- Intensive Care Delirium Screening Checklist (ICDSC)
- Delirium Detection Score (DDS)
- Nursing Delirium Screening Scale (Nu-DESC)
- Neelon and Champagne Confusion Scale.

Postoperative cognitive dysfunction (POCD) is an objective-measured decline in cognitive function including disorders of orientation, attention, perception, consciousness, and judgment persisting beyond the period expected for normal recovery from the physiological and pharmacological effects of anesthesia and surgery.

Elderly patients are at increased risk of POCD, which includes disorders of orientation, attention, perception, consciousness, and judgment. Higher incidence up to 80% has been reported in cardiac surgery patients. In noncardiac surgery patients, up to one-fourth patients can have POCD. The risk factors for POCD include advanced age, lower educational levels, and duration and complexity of surgery. Strategies to prevent POCD include preoperative prehabilitation including physical activity and social involvement, intraoperative optimizing surgical technique to minimize stress and inflammatory response, monitoring and avoiding excessive depth, avoiding hypoxia and hypotension and postoperative early mobilization, avoiding noisy environment, promoting natural sleep cycle, encouraging presence of family members, and use of patient aids such as hearing machine or spectacles, etc.

Use of dexmedetomidine is associated with a lesser prevalence of perioperative neurocognitive disorders

(PNDs) compared to benzodiazepines and propofol. Use of ketamine may rather increase their risk.

Treatment of delirium: All the strategies for delirium prevention should be optimally used. Every attempt should be made to search for the cause of delirium. Delirium can be due to inadequate pain relief, urinal retention, and constipation. Delirium can be a sign of sepsis, inadequate brain perfusion, hypoxia, hypoglycemia, metabolic abnormalities including hyponatremia or uremia, etc.

Sepsis is a potentially dangerous cause of an acute confusional state. One must keep in mind that elderly patients may not demonstrate high fever, tachycardia, or leukocytosis as a response to sepsis. The only manifestation of serious sepsis maybe mental obtundation or confusion.

Pending treatment of cause, pharmacological treatment of delirium should be reserved for hyperactive delirium with potential for injury to the patient or others using minimal doses of dexmedetomidine or haloperidol (ECG for QTc monitoring) or both.

SUGGESTED READING

1. Dauchot PJ, Lina AA. Geriatric anesthesia. In: Brown B (Ed). Risk and Outcome in Anesthesia. Philadelphia: JB Lippincott; 1993. pp. 527-37.
2. Griffiths R, Beech F, Brown A, Dhesi J, Foo I, Goodall, et al. Perioperative care of the elderly. Association of Anaesthetists of Great Britain and Ireland. *Anaesthesia*. 2014;69 (Suppl. 1):81-98.
3. Janis KM. The geriatric patient. In: Kirby R, Gravenstein N (Eds). *Clinical Anesthesia Practice*. Philadelphia: WB Saunders; 1994. pp. 1067-89.
4. Men-it H. The geriatric patient. In: Roger MC (Ed). *Current Practice in Anesthesiology*. St. Louis: Mosby-Year Book; 1990.
5. Miller R, Eriksson L, Fleisher L, Wiener-Kronish J, Cohen N, Young W. *Miller's Anesthesia*, 8th edition. Amsterdam: Elsevier; 2014.
6. Morgan GE, Mikhail MS, Murray M. *Clinical Anesthesiology*, 3rd edition. New York: McGraw Hill; 2002.
7. Sharma G, Goodwin J. Effect of aging on respiratory system physiology and immunology. *Clin Interv Aging*. 2006;1(3):253-60.
8. Tsui BC, Wagner A, Finucane B. Regional anaesthesia in the elderly: a clinical guide. *Drugs Aging*. 2004;21:895-910.

Transurethral Resection of Prostate

Jigeeshu Vasistha Divatia, Shilpushp Jagannath Bhosale, Bindiya Salunke

A 67-year-old male patient who presented with history of dysuria, urgency, and hesitancy is posted for transurethral resection of the prostate (TURP).

Q. What is transurethral resection of the prostate (TURP)?

Ans. Transurethral resection of the prostate remains the surgical gold standard for the treatment of benign prostatic hyperplasia (BPH), which causes urinary obstruction and increases the risk of urinary tract infection. TURP is usually performed in patients in whom the prostate weighs <60 g. TURP involves the resection of benign hypertrophic prostatic tissue by means of a movable electrocautery cutting wire loop located at the end of a urethroscope. The urethroscope is passed through a sheath that has been positioned within the patient's urethra. The prostatic tissue is resected in small strips under direct vision using the diathermy loop, which can both cut and coagulate. The bladder is continuously irrigated with fluid to allow direct vision and to wash away blood and debris. At the end of the procedure, a three-lumen catheter is inserted and irrigation is continued for up to 24 hours after operation. The procedure usually takes 30–90 minutes, depending on the size of the prostate and the level of experience of the operator. It is performed in the lithotomy position.

Q. Describe the anatomy of the prostate gland.

Ans. The prostate gland approximately weighs 20 g and underlies the apex of the male bladder and surrounds the prostatic portion of the urethra. The prostate is formed by enlargement of the urethral glands. A fibrous sheath surrounds the prostate, and the body of the gland consists of a fibromuscular stroma that envelops the glandular tissue. Venous drainage is via the thin-walled veins, or sinuses, of the prostatic plexus.

Although developmentally divisible into two lobes, the prostate gland is anatomically divisible into five lobes.

The median and two lateral lobes of the prostate gland most frequently undergo benign prostatic hypertrophy. The nerve supply to the prostate derives from the prostatic plexus, which originates from the inferior hypogastric (pelvic) plexus. Afferent pain fibers of the prostate, urethra, and mucosa of the bladder originate primarily from sacral nerves 2, 3, and 4 (S2, S3, and S4). Pain impulses from an overstretched bladder travel with sympathetic fibers that have their origin in the 12th thoracic and 1st and 2nd lumbar nerves (T12, L1, and L2). Proprioceptive impulses from the muscular wall of the bladder, which are activated by stretching of the muscular wall as the bladder fills, are carried by the parasympathetic fibers of nerves S2, S3, and S4.

Q. How would you assess this patient for anesthesia?

Ans. Transurethral resection of the prostate is generally performed on elderly men. Hence, one must also take into account multiple comorbidities to which the elderly are prone such as ischemic heart disease (IHD), hypertension (HT), diabetes mellitus (DM), and renal dysfunction. In addition, they may be on treatment for such conditions with medications such as aspirin and oral anticoagulants.

Chronic prostatic hypertrophy may cause obstructive renal insufficiency and urinalysis is mandatory to exclude a urinary tract infection, as if this is left untreated, it increases the risk of postoperative septicemia. A brief discussion of age-related organ changes is given here.

Central nervous system (CNS): Reduced functional tissue in the CNS is probably one of the factors that reduces the anesthetic dose needed in older patients. Old patients often have sluggish, impaired, or absent reflex responses (e.g., pupillary light reflex) which may complicate monitoring

during anesthesia. Loss of function of the special senses such as sight and hearing may lead to apprehension in strange environments, and sometimes sedation is needed to reduce preoperative stress which otherwise can significantly increase sympathetic stimulation. Geriatric population has reduced the ability to generate body temperature and is susceptible to develop hypothermia, particularly during prolonged surgery or the postoperative recovery period. In this context, it is important to remember that core body temperature may differ from peripheral measurements, and the use of esophageal thermometers or infrared thermometers (applied in the auditory) may be preferable to rectal temperature recording.

Peripheral nervous system: Super sensitivity of postsynaptic receptors may prolong the action of muscle relaxants. Interpretation of the significance of poor reflex responses during anesthesia is more difficult in older patients than in the young.

Cardiovascular system: Subclinical and clinical cardiac disease is common in elderly, and impaired cardiovascular function should be expected in elderly patients. Baroreceptor function may be impaired, particularly in patients with chronic congestive heart failure, and the cardiovascular system's ability to compensate for surgical hemorrhage or for the vasodilatory effects of anesthetic agents may be inadequate resulting in severe hypotension. Existing cardiovascular conditions such as congestive heart failure, cor pulmonale, sick sinus syndrome, and the cardiac signs associated with hyperthyroidism should be stabilized before general anesthesia. Anesthetic agents depress cardiac function and cardiac arrest can be precipitated if cardiac arrhythmias are present, particularly ventricular arrhythmias that are refractory to therapy, right bundle branch block, and bradycardia. Monitoring the heart rate and systolic and diastolic blood pressures (BPs) by direct or indirect methods is desirable throughout anesthesia as is ECG (electrocardiogram) recording, monitoring pulse character, and regular examination of visible mucous membranes for capillary refill time.

Respiratory system: Age-related degenerative changes progressively decrease pulmonary function and physical changes occur in the lungs and the chest wall. There is reduced alveolar surface area and diffusion capacity, pulmonary fibrosis, reduced lung elasticity, and reduced mechanical ventilation reserve. Chronic obstructive lung disease is common. All of these changes impair gaseous exchange during anesthesia, hence oxygen supplementation is beneficial, and in some cases, the use of bronchodilators may be indicated. Pulmonary embolism is a common postoperative complication in old people.

The incidence of deep vein thrombosis is reported to be as high as 45% in those aged over 40 years and 65% in patients aged over 71 years.

Renal: Impaired renal function prolongs the plasma half-life of drugs eliminated via the kidney and may alter fluid, electrolyte, and acid-base balance, so screening for renal function is important before anesthesia. It is good practice to administer a balanced solution intravenously before and during anesthesia to facilitate control of fluid, electrolyte, and acid-base balance and to maintain renal perfusion. If hypotension occurs during anesthesia, tubular ischemia may result leading to acute tubular necrosis and renal failure. Advancing age and general anesthesia are both important risk factors for the development of acute renal failure. Other risk factors include the administration of nonsteroidal anti-inflammatory drugs (NSAIDs), aminoglycosides, tetracyclines, and other nephrotoxic drugs.

Liver: Most liver function tests usually remain normal in geriatric patients and this probably reflects the huge reserve capacity of this organ; however, in humans, bromsulphalein (BSP) retention does increase with age. In the presence of impaired hepatic function, the plasma clearance rate for drugs may be decreased resulting in increased duration of action. At the same time, drugs and nutrients that need to be converted to an active form by the liver may exhibit reduced activity.

Q. What is the position of the patient for TURP? Describe the complications associated with that position.

Ans. Patient position during TURP: An important aspect during TURP is the position given to the patient. For optimal surgical exposure, the patient is put in lithotomy position. The first consideration is to fit the equipment to the patient and not the patient to the equipment.

How to give lithotomy position: Patient is positioned supine on the table, anesthesia instituted and then patients' legs are elevated together, flexed together, and only then put in stirrups. Hence, the thighs are flexed at about 90° to the abdomen and outwardly rotated. For TURP, lithotomy with obtuse angle between the thigh and the abdomen is desirable.

Variations of lithotomy position:

- Lithotomy with Trendelenburg position
- Walcher's position
- Exaggerated lithotomy position (Trendelenburg position with sheets/towels beneath the sacrum to tilt it up).

Complications of the lithotomy position:

- *Venous stasis:* It occurs in prolonged lithotomy. Stasis occurs at points of compression by equipment or at the

groin due to thigh flexion. Patients with varicose veins are at high-risk:

- *Preventive measure:* If lithotomy position is for >15 minutes, then the legs have to be protected by elastic stockings.
- *Peripheral nerve injury:* It occurs either due to direct trauma or usually due to compression of blood vessels supplying the nerve:
 - *Obturator nerve (L2, 3, 4):* It leaves the pelvis through the obturator foramen and starts dividing in the upper thigh. Thus, acute flexion of the thigh to the groin causes nerve compression and trauma. This leads to weakness or paralysis of adductors of thigh. This leads to difficulty in walking and will manifest as an outward swing of the leg. The obturator nerve can be electrically stimulated during surgery, causing a vigorous movement of the leg called the obturator nerve reflex.
 - *Saphenous nerve (L2, 3, 4):* Compression of the medial aspect of the leg against the knee brace results in sensory loss to the medial side of the thigh. Femoral nerve is injured by acute thigh flexion with angulation against the undersurface of the pubic ramus.
 - *Common peroneal nerve (L4, L5-S1, S2):* It courses around the head of the fibula. It becomes flattened here and is prone to pressure injury to its nutrient vessel. Any prolonged compression of the lateral aspect of the knee can result in nerve injury and leads to foot drop.
- *Cardiorespiratory compromise:* The lithotomy position results in an 18% reduction in the vital capacity, which is further increased by Trendelenburg tilt (up to 14.5% with a 20° tilt). Spinal anesthesia (SA) causes pooling of blood in the lower limbs, and this may be abruptly returned to the central circulation when the legs are raised to give the lithotomy position. All this may result in precipitation of respiratory and/or cardiac failure in elderly patients with preexisting disease and diminished cardiopulmonary reserve.

Q. What is the primary concern and complication associated with TURP?

Ans. The primary concern associated specifically with TURP is intravascular absorption of large volumes of irrigating fluid during the procedure. The absorption occurs predominantly through exposed venous sinuses of the surgical capsule. Hypoosmolality is more important than hyponatremia in causing CNS disturbances.

Complications can be categorized according to the time of occurrence.

Intraoperative complications:

- TURP syndrome.
- *Hypothermia:* This is due to use of cold irrigation fluids.
- *Myocardial infarction:* The incidence of perioperative myocardial infarction ranges from 1 to 3%.
- Bleeding and bladder perforation are also common occurrences during TURP.

Postoperative complications:

- TURP syndrome
- Clot retention
- Bleeding and disseminated intravascular coagulation
- Postoperative cognitive dysfunction.

Q. What are the first signs and symptoms of TURP syndrome?

Ans. Approximately 1–8% of TURP surgeries may have TURP syndrome. Recent larger studies have demonstrated lower incidence rates of between 0.78% and 1.4%, with mortality rates of 0.2–0.8%. The development of TURP syndrome can be from 15 minutes of procedure to 24 hours postprocedure. The clinical manifestations brought about by intravascular fluid absorption are referred to as the TURP syndrome, and the degree of symptoms depends on the type, magnitude, and extent of absorbed fluid. The anesthesiologist must recognize signs and symptoms of developing TURP syndrome. For the patient undergoing TURP under regional anesthesia (subarachnoid block or epidural block), the first sign has been described classically as restlessness and mental confusion. However, presentation is variable, and the syndrome may manifest initially with nausea, vomiting, dizziness, headache, unresponsiveness, or transient visual changes. Hemodynamic instability (HT, hypotension, heart rate changes, cardiac arrhythmias, pulmonary edema, or cyanosis) may be the first indication of a development of TURP syndrome.

Q. Which is the best irrigating fluid for TURP? Give a brief description of all irrigating fluid used for TURP.

Ans. Several types of fluids are available for use during a TURP procedure. But the ideal irrigant should be:

- Isoosmolar and nonhemolytic
- Nonelectrolytic
- Nontoxic
- Transparent
- Nonmetabolizable
- Rapidly excretable
- Inexpensive and sterile.

Irrigating solutions available are:

Saline and lactated Ringer's solution: It conducts the electrical current but not the thermal properties of the electrostatic unit (ESU). They disperse the electrical current, and no cutting or coagulation occurs. Electrolyte-based

solutions are therefore not used as an irrigating solution for TURP procedures.

Water: Water may be used for short TURP procedures, but it is not used commonly because it is hypotonic and eventually will result in hemolysis; releasing free serum hemoglobin and potassium. Although a serum hemoglobin level is not toxic until it reaches 600 mg, there is a risk of serum hemoglobin combining with abnormal serum proteins, rendering it nephrotoxic. Release of potassium can lead to hyperkalemia, resulting in cardiac arrhythmias and muscular weakness. Severe hyponatremia and hyposmolality can occur with CNS symptoms including convulsions and coma.

Glucose: Glucose 5.4% has been used in the past but is no longer used because it causes tissue charring and also leaves the surgical site and instruments very sticky. This makes the procedure surgically difficult. Additionally, elevated serum glucose levels can occur as the fluid is absorbed into the bloodstream. This poses an unnecessary risk for patients with diabetes.

Urea: Urea 1.8% also has been used in the past but is no longer used as it crystallizes on the instruments. Urea is permeable to the intracellular and extracellular spaces, which results in elevated serum urea levels even when only small amounts are absorbed. Elevated serum urea levels result in nausea, vomiting, headache, tachycardia, elevated blood pressure, diminished vision, convulsions, and coma. Urea also has an osmotic diuretic effect, so dehydration can occur.

Sorbitol: Sorbitol 3.3% (osmolality: 165 mOsm) is used infrequently as an irrigation solution. It is metabolized into carbon dioxide, glucose, and water. The kidneys excrete all three substances as by-products. Water intoxication remains a problem when large quantities of sorbitol are absorbed. Additionally, the resultant elevated serum glucose levels can pose problems, especially for patients with diabetes. It may also cause lactic acidosis.

Mannitol: Even though mannitol 3% is an osmotic diuretic, it has been used for irrigation. Dehydration, hyperosmolality, and acidosis with resultant neurological disturbances can occur.

Crystal: It is a combination of sorbitol 2.7% and mannitol 0.54%. It is not widely used. Sorbitol is metabolized to fructose, which may present problems to patients with hypersensitivity to fructose.

Glycine: Currently, the most commonly used irrigant is 1.5% glycine. Glycine is hypoosmolar at 220 mmol/L, is nonconductive and nonhemolytic, and has a neutral visual density.

The liver metabolizes glycine into ammonia, water, and glycolic acid, resulting in hyperammonemia. When large amounts of glycine are absorbed, hyperammonemia and water intoxication can lead to cerebral edema and seizures. Increased absorption can also cause hyperoxaluria and hypocalcemia (glycine is metabolized to glyoxylic acid and oxalic acid; the latter forms calcium oxalate crystals in the urinary tracts and may contribute to renal failure).

Q. What is TUR syndrome? Describe the physiology and management of TUR syndrome.

Ans. Transurethral resection syndrome is a constellation of symptoms and signs caused primarily by absorption of irrigating fluid. The factors leading to this syndrome have been discussed above. The features of the syndrome include:

- CNS manifestations are disorientation, restlessness, confusion, agitation, drowsiness, convulsions, and coma. This is usually due to water intoxication, dilutional hyponatremia, and hyposmolality. Blindness, delayed recovery from anesthesia, and drowsiness may be due to glycine toxicity.
- Cardiovascular system manifestations are dyspnea, pulmonary congestion, pulmonary edema, and cardiac arrest. This is due to fluid overload and negative inotropic effect of hyponatremia.
- Hemolysis.
- Hypothermia—bladder irrigation with room temperature irrigation fluids can drop body temperature $>2^{\circ}\text{C}$.

Factors affecting intravascular absorption and methods to reduce the risk of the TURP syndrome are as follows:

- Number and size of the venous sinuses opened
- *Duration of exposure:* Reduction of the operative time to <1 hour decreases the chance of TUR syndrome. The rate of absorption of fluid is 10–30 mL/min.
- Venous pressure at irrigant–blood interface.

The irrigating fluid bag should be at a maximum height of 60 cm (2 feet) above the level of the pubic symphysis. This leads to a hydrostatic pressure of 60 cm of water which is maximum allowable.

Intravascular absorption of irrigating fluid can lead to:

- *Overhydration:* It was first described as a syndrome in 1973 when 1.2% glycine was used.

The syndrome of overhydration consists of a triad of signs:

- Bradycardia
- Elevated systolic and diastolic BPs with increased pulse pressure
- Cerebral agitation and depression.

Cerebral signs may be variable but generally commence with headache, dizziness, restlessness, agitation, and confusion graduating to progressive obtundation, stupor, and coma. Neuromuscular disturbances ranging from

twitches to seizures can occur. Later dyspnea, cyanosis, refractory hypotension, and cardiac arrest may follow.

The absorption of large amounts of electrolyte free irrigating solution results in increased intravascular volume, hemodilution, hyponatremia and development of left heart failure, and pulmonary and cerebral edema and ultimately cardiovascular collapse. It is seen that the faster the fall of serum sodium levels, the more are the toxic symptoms likely to develop. A drop of 20–30 mEq/L or an absolute value of 120 mEq/L indicates a severe reaction. Acute hyponatremia is produced initially by the dilutional effect of a large volume of absorbed irrigation fluid, but later is caused by natriuresis, and may cause headache, altered level of consciousness, nausea and vomiting, seizures, coma, and death. Hypoosmolality is more important than hyponatremia in CNS disturbance. The Nernst equation predicts that a moderate decrease in extracellular sodium concentration only minimally alters membrane excitability. Patients who are hyponatremic but have a normal osmolality are likely to be asymptomatic. Rapid reduction in plasma osmolality overwhelms neuronal compensatory mechanisms. Free water is absorbed into the brain parenchyma, causing water intoxication, cerebral edema, and raised intracranial pressure.

In less florid cases, hypothermia, weight gain, and postoperative diuresis are seen. Unusual symptoms of fluid absorption include visual disturbances ranging from blurred vision and papillary dilatation to transient edema. These symptoms were ascribed to cerebral and spinal edema, respectively.

When overhydration syndrome is suspected, surgery should be stopped and immediate measurement of serum sodium and serum osmolality should be done. Those patients with isolated alteration of sodium concentration may not develop serious sequelae and spontaneously diurese and need no further treatment. When a patient develops neurological signs, prompt intervention is required. Treatment is directed toward reversing the flow of water into the cells and correcting hypotonicity.

TABLE 1: Serum sodium systemic effects.

S. Sodium levels	Central nervous system effects	Electrocardiogram effects
120 mEq/L	Confusion and restlessness	Wide QRS
115 mEq/L	Somnolence and nausea	Wide QRS and ST elevation
110 mEq/L	Seizures and coma	Ventricular tachycardia/fibrillation

The patient is to be oxygenated and diuretic such as furosemide is to be administered with restriction of fluid intake. Then, correction of hyponatremia is best done by

hypertonic saline in which case sodium requirement is calculated based on total body water. The aim should be to bring the sodium level >120 mEq/L. As administration of hypertonic saline is not without the risk of fluid overload, simultaneous furosemide should be administered. Therapy should be monitored by assessing clinical and biochemical improvement (**Table 1**). Serum sodium concentration should be measured in conjunction with serum osmolality. If the osmolality is normal or nearly so, no intervention is recommended to correct serum sodium if the patient is asymptomatic. Rapid correction of hyponatremia may lead to central pontine myelinolysis (CPM). The presence of symptoms has been described as the most important factor determining morbidity and mortality from hyponatremia. Dilutional hyponatremia may prolong the action of nondepolarizing neuromuscular blocking agents, and may cause broadening of the QRS complex or T-wave inversion.

If measured serum osmolality is <260 mOsmol/kg, aim to raise Na^+ by no more than 10–12 mmol/24 hours).

Q. Enumerate complications of TURP? Give brief description of each.

Ans. Apart from TURP syndrome (described above), other complications of TURP are described below:

Glycine toxicity: Intravascular absorption of glycine from the irrigant can cause direct neurotoxic effect. It is a major inhibitory neurotransmitter in the spinal cord and midbrain. Glycine may lead to encephalopathy and seizure via N-methyl-D-aspartate (NMDA) receptor activity (an excitatory neurotransmitter). NMDA receptor activity is markedly potentiated by glycine and, along with its role as an inhibitory transmitter, may facilitate excitatory transmission in the brain through an allosteric activation of the NMDA receptor. Signs of glycine toxicity are nausea, vomiting, headache, malaise, weakness, and visual symptoms which range from blurred vision to complete blindness (glycine is now thought to be the most likely cause of visual disturbances). Glycine also causes renal toxicity. It is broken down by the liver (oxidative deamination) to ammonia and oxalate. This oxalate excretion continues long enough postoperatively for calcium oxalate to deposit in the kidneys and cause renal failure. Glycine toxicity can be treated with arginine which increases ammonia metabolism via the urea cycle. Magnesium exerts negative control on the NMDA receptor. Therefore, a trial of magnesium therapy for seizure control deserves consideration, especially if osmolality is near normal. The normal value of serum glycine is 13–17 mg/L. Vision approaches normal within 24 hours as glycine levels return to normal. This is predictable as the half-life as glycine is 85 minutes. Thus, reassurance may be the best treatment. Glycine may also directly depress the myocardium. N-methyl-D-aspartate receptor activity is

potentiated by glycine, which paradoxically may precipitate encephalopathy and seizures. Magnesium (whose plasma level may also be reduced through dilution) exerts a negative control on the NMDA receptor and also having a membrane-stabilizing effect, and magnesium therapy should be considered as part of the therapy for seizures in TURP syndrome.

The liver and kidneys metabolize glycine by oxidative deamination to glyoxylic acid and ammonia. The redistribution half-life of glycine is 6 minutes. The terminal half-life of glycine is dose-dependent and varies from 40 minutes to several hours. The role of hyperammonemia during TURP syndrome remains unclear, although ammonia is considered to be a cerebral depressant:

- **Hemolysis:** Danger of hemolysis exists when hypotonic fluids are used. It leads to hemoglobinemia, hemoglobinuria, anemia, and hyperkalemia. Acute renal failure can occur due to free hemoglobin and other breakdown products. Patient develops chills, clamminess, chest tightness, rising BP, and bradycardia. The treatment is aimed at promoting diuresis and correction of biochemical and hematological anomalies.
- **Blood loss:** It is difficult to quantify. Visual estimation is difficult due to dilution of blood with the irrigating fluid. Also, the warning signs such as tachycardia and hypotension are masked by bradycardia and HT of overhydration syndrome. A rough estimate is 15 mL/g of resected prostatic tissue or 2.6–4.6 mL/min of resection time. Methods of quantifying are:
 - Radioactive labeling of red blood cell (RBC) or albumin
 - Alteration in electrical conductivity
 - Calorimetric method.

The way to calculate true blood loss is to collect all of the irrigant–blood mixture and to measure its hematocrit: Blood loss (mL):

$$= \frac{\text{Hb of irrigating fluid (g/mL)}}{\text{Patient's Hb (g/L)}} \times \text{Amount of irrigating fluid}$$

Causes of increased blood loss: Blood loss during TURP is inevitable and is typically in the region of 500 mL. It is difficult to quantify due to the large volume of irrigating solution used. Patients lose between 2.4 and 4.6 mL of blood per minute of resection, no matter whichever anesthetic technique is used. In theory, blood loss can be estimated by assaying the hemoglobin concentration of the discarded irrigation fluid by measuring the electrical conductivity of the discarded irrigation fluid, or in the laboratory by radioactive albumin or red-cell labeling techniques. Visual assessment of the color of the discarded irrigation fluid is unreliable.

Factors associated with excessive bleeding include a large gland, extensive resection (>40–60 g of prostate chippings), coexisting infection, prolonged surgery (>1 hour), and the presence of a preoperative urinary catheter. The histology of the gland is not associated with differences in bleeding. In practice, frequent measurement of the hemoglobin is the most useful investigation.

Urokinase released from raw prostate tissue may provoke systemic fibrinolysis, which may worsen postoperative hemorrhage. Where blood loss is extensive, a bolus of tranexamic acid, e.g., 15–25 mg kg⁻¹, may reduce the volume of hemorrhage.

Circulatory overload: Due to fluid absorption or iatrogenic overinfusion leading to increased venous pressure and thus bleeding.

Vasodilatation: Due to SA, lithotomy position, dependent position of operative area, favors blood pooling and raised venous pressure in the pelvis and prostatic sinuses. Shivering, coughing, and straining can also increase venous pressure.

Infection: Indwelling catheter will cause local tissue congestion and hyperemia leading to increased bleeding.

Excessive bleeding may result from the activation of plasminogen by urokinase (plasma protein fraction liberated from prostatic tissue) to plasmin causing fibrinolysis. This leads to generalized oozing from the prostatic fossa, delayed postoperative bleeding, bleeding from puncture sites, etc. This is treated with e-aminocaproic acid (EACA), tranexamic acid, fibrinogen, or blood.

Bleeding can be decreased by using cooled irrigants and by selective cooling of pelvic organs. However, the risk of hypothermia should be considered in the elderly.

- **Perforation:** This can be—
 - Urethral
 - Intraperitoneal (rupture of dome)
 - Extraperitoneal (rupture at neck)
 - Capsular or periprostatic extravasation.
- **Causes:** It may be due to instrumentation or overdistension of bladder.
- **Signs and symptoms are as follows:**
 - Abdominal distension
 - Patient may complain of abdominal pain which may be generalized, suprapubic, or radiating to the shoulder. In case of SA (level <T10), capsular tear will cause suprapubic pain
 - Rigidity
 - Autonomic signs such as pallor, nausea, vomiting, and diaphoresis
 - Bradycardia, HT, and shock-like picture with large extravasation
 - Nonreturn of irrigating fluid.

Treatment: Localized, extraperitoneal, periprostic, or urethral tears are treated with catheterization and antibiotics. More extensive tears require exploration and repair of perforation and drainage of perivesical space. Intraperitoneal rupture requires surgical treatment and extraperitoneal rupture is treated conservatively.

- **Hypothermia:** The common causes are illustrated below—
 - In regional anesthesia, there is redistribution of body heat from deep to superficial tissues in the area of the block.
 - In the area of block, ability to shiver is lost and metabolic response to trauma is obtunded.
 - There is reduced thermoregulatory capacity in elderly patients.
 - Frequent use of cold intravenous fluids (IVF) and irrigating fluids.
 - Patients are on cold operation theater (OT) tables with cool preparatory solutions.
 - Cold and dry operating room.
- **Bacteremia:** During the procedure, bacteria from the infected urinary tract may enter bloodstream. Patient presents with rigors and hypotension.

Q. Describe anesthetic management of TURP. What is the preferable anesthetic technique and why?

Ans.

- **Anesthesia management:** Ideal anesthetic should provide adequate operative conditions and analgesia. Physiological disturbance should be minimal and compensatory mechanisms should not be compromised. Bleeding must be minimized. There should be adequate muscle relaxation to provide good irrigant flow into the bladder and to allow good surgical access. Doses of the drugs must be carefully adjusted to patient's requirement. The technique must allow early detection of overhydration, perforation, and hemolysis.
- **Subarachnoid block is the method of choice as:**
 - Little anesthetic is required.
 - Patient remains awake and can be assessed regularly.
 - Physiologic disturbance is minimal.
 - Respiration is affected to the minimum.
 - Early recognition of complications (level to be restricted to T10).
 - Peripheral vasodilatation helps reduce circulatory overload.
 - Morbidity and mortality is low.

Note the nerve supply of the prostate gland and bladder neck:

- Sympathetic—T11–L2, L3
- Parasympathetic—S2–S4
- Spinal levels of pain conduction—T11–L2, S2–S4

- There is little evidence to demonstrate significant difference in outcomes between general and SA. SA may offer several advantages over general anesthesia. Early recognition of capsular tears and bladder perforation is also possible as the patient complains of periumbilical or shoulder pain provided the spinal level is limited to T10.
- SA has traditionally been avoided in patients with ischemic heart disease; however, the incidence of myocardial ischemia is the same for spinal and general anesthesia. A spinal block to T10 is required to eliminate the discomfort caused by bladder distension: 2.5–3.0 mL of 0.5% plain or hyperbaric bupivacaine may be used.

The lithotomy position in combination with a head-down tilt reduces the tidal volume and the functional residual capacity, and increases the likelihood of gastric regurgitation. Tracheal intubation and positive pressure ventilation may counteract these problems. However, the use of a laryngeal mask airway with spontaneous ventilation in selected patients is also an acceptable alternative.

Q. When is TURP syndrome likely to occur?

Ans. The time to onset of TURP syndrome depends on numerous factors, including the experience of the surgeon, the surgeon's aggressiveness with the electrocuting loop, the pathology and size of the gland, the amount of tissue removed, and the amount of irrigation used. The incidence of morbidity increases as resection time exceeds 60 minutes, and for many years, it was believed that TURP syndrome was unlikely during the first hour of resection. However, TURP syndrome can also develop more rapidly.

The patient is not free of risk once the resection is completed. If the integrity of the prostatic capsule or wall of the bladder is violated during surgery, irrigating fluid may be sequestered in the intraperitoneal and extraperitoneal space during resection. The fluid may be absorbed into the intravascular space during the postoperative period and results in intravascular fluid overload and symptoms of TURP syndrome.

Q. What is the treatment for TURP syndrome?

Ans. Treatment of TURP syndrome should begin the moment the problem is recognized:

- Terminate surgery as quickly as possible and switch to normal saline for continuous bladder irrigation. Be sure that the irrigation is warm, as the bladder irrigation now should be to prevent the development of hypothermia.
- Treatment should involve supporting respiration (if necessary, with intubation and ventilation) and the circulation. Bradycardia and hypotension should be

treated with atropine, adrenergic drugs, and IV calcium. IV anticonvulsants (e.g., diazepam or lorazepam) should be used to control seizures and IV magnesium therapy is considered, if seizures prove to be difficult to control. Obtain the following baseline laboratory tests: complete blood count, platelet count, electrolytes, and clotting studies, if a bleeding problem is suspected. Prothrombin time and/or international normalized ratio (INR), partial thromboplastin time, and fibrinogen level should be included in the coagulopathy work up.

- Administration of intravenous normal saline and diuretics may be all that is needed to correct the problem. Administer injection furosemide, 20 mg, intravenously. If the patient is on chronic diuretics, a dose of 40 mg or more may be required, but dosing should be based on the diuresis obtained initially from 20 mg bolus 15%. Mannitol can also be used as an alternative to furosemide. Maintain intravascular volume with normal saline as diuresis progresses.
- If the patient demonstrates significant effects from hyponatremia, intravenous administration of hypertonic saline may be appropriate. Restrict the use of hypertonic saline to patients who have developed central seizures or cardiac dysfunction.
- Consider placement of a central venous catheter to guide fluid replacement during the immediate postoperative period.
- If hemodynamic instability develops, consider placement of an arterial catheter and pulmonary artery catheter to aid in resuscitation.
- Monitor the serum potassium level. Patients frequently become hypokalemic as diuresis occurs.
- Reassure patients that any symptoms, especially visual changes, are only temporary and that their symptoms will dissipate as their condition improves.

Q. Why not replace sodium deficit with hypertonic saline in patients suffering from TURP syndrome?

Ans. The use of hypertonic saline for correction of hyponatremia associated with TURP syndrome should be restricted to patients demonstrating significant symptoms, namely, central seizures or cardiac dysfunction due to electrolyte imbalance. If hypertonic saline is chosen for fluid replacement, close attention must be paid to the patient's electrolyte and intravascular fluid status. Remember that the patient has not lost sodium, but has gained water. Excessive administration of hypertonic saline results in additional fluid overload and complicates an already difficult management problem. Hypertonic saline should be administered through a central line at a rate no greater than 100 mL/h.

Q. Is it possible to calculate how much irrigating fluid has been absorbed?

Ans. The amount of irrigating solution absorbed can also be estimated by comparing sodium levels at any time during the procedure with levels at the start of the procedure:

$$\text{Volume absorbed} = \frac{\text{Preoperative S.Na}}{\text{Postoperative S.Na}} \times \text{ECF} - \text{ECF}$$

Example:

A 70-kg man undergoes TURP under subarachnoid block (spinal anesthetic). After 50 minutes of resection, he complains of headache and appears somewhat disoriented. The procedure is immediately terminated, and a blood sample is sent to the laboratory for electrolyte analysis. The patient's preoperative serum sodium concentration was 142 mEq/L compared with the immediate postoperative value of 106 mEq/L. If the patient has an extracellular fluid (ECF) compartment of approximately 20% of body weight, his ECF volume at the start of the procedure was about 14 L ($0.20 \times 70 = 14$ L). Using the above formula, extracellular volume has increased to $(142/106) \times 14$ L = 18.75 L. Subtracting his initial extracellular volume of 14 L from the postoperative extracellular volume of 18.75 L yields an absorption of 4.75 L. This figure represents a minimal volume of absorption, because any fluid that has shifted into the intracellular space is lost in the calculation.

A more accurate technique for calculating fluid absorption is to add 1% ethanol to the irrigating solution and then to monitor and quantify the amount of ethanol that the patient expires. This method is sensitive enough to detect approximately 75 mL of fluid absorption per 10 minutes of surgery.

1 L uptake of fluid within 1 hour roughly corresponds to decrease in the serum sodium concentration of 5–8 mmol/L. Risk of circulatory overload occurs when the weight of the gland is >45 g.

Q. What can be done to minimize the risk of developing TURP syndrome?

Ans.

- The patient must be prepared properly for surgery. Preparation should include adequate hydration, electrolyte analysis, and coagulation profile. Patients who are debilitated and demonstrate poor reserve may benefit from placement of hemodynamic monitors for preoperative assessment and optimization as well as for intraoperative monitoring.
- The most important step in minimizing the risk of TURP syndrome is to limit the duration of surgery. Because fluid can be absorbed at a rate >50 mL/min, it is possible to place nearly 3 L of fluid into the intravascular and interstitial spaces within 1 hour of resection time. Limit resection time to 1 hour or less.

- Irrigation fluid is absorbed at a rate of between 10 and 30 mL/min of operating time. 5–20% of patients will absorb >1 L. In some centers, ethanol 1% is added to the irrigation solution and the patient's breath is tested for ethanol every few minutes: a positive test indicates a significant quantity of fluid has been absorbed.
- The hydrostatic pressure created by the fluid irrigating the surgical site must be minimized. Because the irrigating fluid flows by gravity, the bag of irrigation should not hang higher than 60 cm above the operative field.
- The surgeon should limit the extent of bladder distention created by the irrigant. Frequent drainage of the bladder by the surgeon reduces the amount of irrigant absorbed.
- Careful surgical resection minimizes exposure of the venous sinuses and preserves the capsule of the prostate. Hence, operator experience plays a major role in the prevention of TURP syndrome.
- Blood pressure must be stable. A decrease in pressure lowers the periprostatic venous pressure and allows increased absorption of fluid.
- Intraprostatic vasopressin injection shown to reduce bleeding and absorption of irrigating fluid.
- Use of newer techniques such as bipolar electrode resection, transurethral vaporization or needle ablation, and laser excision of prostate (Holmium: yttrium-aluminum-garnet and potassium titanyl phosphate lasers) can minimize the risk of bleeding and also absorption of irrigating fluid, practically eliminating the risk of TURP syndrome.

Newer techniques of prostatic resection use different types of energy (heat, laser, ultrasound, or microwave) to vaporize prostatic tissue and coagulate surrounding blood vessels. These techniques are reported to cause less hemorrhage than conventional TURP, but specimens for histology cannot be obtained. Since diathermy is not used, normal saline may be used as the irrigating solution, minimizing the risk of the TURP syndrome.

Q. What precautions should be taken for TURP in patients with permanent pacemaker?

Ans. Electromagnetic interference in pacemakers has often been reported in association with the cutting mode of monopolar electrocautery and sometimes even in association with the coagulation mode.

Surgical diathermy assimilates current in the radiofrequency range of 500–2,500 kc/s. This current enters the patient as the active electrode passes through the tissues, and then returns to the diathermy unit via the large indifferent electrode. The use of properly grounded instruments and placing the large indifferent electrode low on the body of the patient can minimize but do not

eliminate the hazards of electrocautery. The electric current is not limited to the tissues interposed between the two electrodes of diathermy, but spreads out and penetrates the entire body of the patient. This stray current may be detected by the demand pacemaker, and can be interpreted as spontaneous cardiac activity causing inhibition of the pacemaker function.

The grounding pad should be placed in such a position that the direction of current flow is at right angles to the pacemaker lead. This decreases the chance of electromagnetic coupling leading to current generation within the lead.

Detailed knowledge of pacemaker nomenclature and the significance of the individual functions and the response to magnet should be confirmed. Other measures for temporary pacing such as transvenous pacing, external pacing, and defibrillation should be kept available. Normally VVI (on demand ventricular) pacemakers revert to VOO (asynchronous continuous ventricular pacing) mode during electrical interference or can be done with the magnet placed on pulse generator, but DDD pacemakers do not automatically revert to DOO (asynchronous dual chamber pacing) and may respond to the magnet by being reprogrammed. Cardiac function should be monitored continuously by means of ECG recording and pulse oximetry monitoring.

Q. How do you manage patients on anticoagulants posted for TURP?

Ans. A significant number of patients posted for TURP have comorbidities such as myocardial infarction and stroke, congestive heart failure, atrial fibrillation, mechanical heart valves, deep vein thrombosis, significant vascular disease, and diseases associated with vascular disease such as diabetes and would be on anticoagulants and/or antiplatelets for the reduction of thromboembolic risks.

These risks include arterial and venous thromboembolism, major adverse cardiac and cerebrovascular events, and more specifically stroke, pulmonary embolus, and major cardiac adverse events which can and usually are far more life-altering than hemorrhagic complications.

American Urological Association (AUA) suggests that no elective procedure should be performed in patients within 1 year after the placement of bare-metal or drug-eluting stent. Antiplatelet therapy continues to be the principal treatment to reduce the risk of thrombosis of coronary stents or coronary artery bypass grafts (CABGs). Primary and secondary prevention and treatment of IHD, postcoronary stenting, or CABG include dual antiplatelet therapy of aspirin with a P2Y₁₂ inhibitor, most commonly, clopidogrel. Dual antiplatelet therapy should be given for at least 12 months in patients with recent acute coronary syndrome treated with either CABG or percutaneous coronary stenting.

For TURP surgery, low-dose aspirin should be continued perioperatively in patients with cardiac risk factors.

Vitamin K antagonists such as warfarin have been the mainstay of therapy in secondary stroke prevention for atrial fibrillation and venous thrombosis prevention till the introduction of direct oral anticoagulants (DOACs) such as indirect factor Xa inhibitors such as rivaroxaban, apixaban, and direct thrombin inhibitors such as dabigatran.

Direct oral anticoagulants are novel anticoagulants with many advantages such as reduced need for monitoring of therapy, shorter therapeutic half-life, and fewer major bleeding complications compared with warfarin. DOACs have disadvantages such as a lack of reversal agents for factor Xa inhibitors; however, recently idarucizumab has been approved by the US Food and Drug Administration (FDA) for the reversal of dabigatran.

Newer techniques such as holmium or thulium laser enucleation (HoLEP/ThuLEP) or GreenLight photovaporization of the prostate (PVP) should be considered in patients who are at higher risk of bleeding, specifically those on anticoagulants drugs.

SUGGESTED READING

1. Gravenstein D. Transurethral resection of the prostate (TURP) syndrome: a review of the pathophysiology and management. *Anesth Analg.* 1997;84(2):438-46.
2. Hahn RG. Fluid absorption in endoscopic surgery. *Br J Anaesth.* 2006;96(1):8-20.
3. Hahn RG. Relations between irrigant absorption rate and hyponatraemia during transurethral resection of the prostate. *Acta Anaesthesiol Scand.* 1988;32(1):53-60.
4. Hanson RA, Zornow MH, Conlin MJ, Brambrink AM. Laser resection of the prostate: implications for anesthesia. *Anesth Analg.* 2007;105(2):475-9.
5. Hawary A, Mukhtar K, Sinclair A, Pearce I. Transurethral resection of the prostate syndrome: almost gone but not forgotten. *J Endourol.* 2009;23(12):2013-20.
6. Kluger MT, Szekely SM, Singleton RJ, Helps SC. Crisis management during anaesthesia: water intoxication. *Qual Saf Health Care.* 2005;14(3):e23.
7. Olsson J, Nilsson A, Hahn RG. Symptoms of the transurethral resection syndrome using glycine as the irrigant. *J Urol.* 1995;154:123-8.
8. O'Donnell AM, Foo ITH. Anaesthesia for transurethral resection of the prostate. *CEACCP.* 2009;9(3):92-6.

Proximal Fracture Femur

Jigeeshu Vasistha Divatia, Bhakti Trivedi, Debashree Lahiri

A 72-year-old male with history of controlled hypertension (for 12 years) and diabetes mellitus (for 15 years) taking atenolol 25 mg BD, glimepiride 2 mg HS, and ecosprin 75 mg presents to the emergency department (ED). After lunch, he slipped in the bathroom, which resulted in swelling of the right hip area with excruciating pain. He was rushed to the casualty by his son at 10 PM (within 1 hour of fall).

Q. What are the types of hip fractures?

Ans. The most common three types of hip fractures are:

1. *Fracture neck femur:* It occurs in the intracapsular part between below the head of femur, in the neck of the femur (may lose blood supply to the head of femur). It may be displaced or undisplaced. Displaced fracture will need total hip replacement (THR) and undisplaced may not need hip replacement (but usually operated on with pin fixation).
2. *Intertrochanteric fractures*—fractures occur between greater and lesser trochanter.
3. *Subtrochanteric fractures*—fractures occur below the lesser trochanter or further down.

Q. Enumerate the goals of management of proximal fracture femur.

Ans. Transfer of the patient to the hospital by ambulance should be after immobilization at the site of injury. Strategies for analgesia, fluid therapy, patient warming, and pressure care therapy during emergency room stay should be in place. Early fracture fixation after appropriate medical optimization should be done. Regional anesthesia is preferred. Postoperative care and rehabilitation program extending in the posthospital discharge period should be the norm. There should be a “fast-track” system for the management of proximal fracture femur cases coming into the emergency department. It has been found that streamlining flow through the emergency department of these elderly patients with hip fracture, who are at risk of significant morbidity and mortality, leads to prompt surgical fixation of the fracture and effective rehabilitation. A protocol-driven multidisciplinary approach leads to faster

onset of definitive management. This is of importance as surgery is the best analgesic for these patients.

Q. How would you assess a patient with proximal fracture femur preoperatively?

Ans. Preoperative assessment by the anesthetist is mandatory, though now more and more orthogeriatricians are also involved in the assessment. This allows for preoperative optimization of the patients along with anesthetic planning and risk assessment.

Comorbidities: Due to preponderance of elderly patients, there is a high chance of multiple comorbidities which need to be evaluated in the preoperative period (Refer to the chapter on *Geriatrics*).

It should also be noted that the elderly patients are on multiple drugs and this may lead to increase chance of adverse drug reaction. Hence, it is necessary to review the medication list and identify pharmacokinetic and pharmacodynamic interactions.

Q. What investigations are necessary?

Ans. Complete blood count: Preoperative anemia is common due to fracture-related hemorrhage, hemodilution, poor nutrition and/or chronic disease. It has been suggested that older patients require a higher blood transfusion trigger than is generally applicable for patients undergoing elective surgery, to the extent that preoperative transfusion should be considered if Hb is <9 g/dL, or if Hb is <10 g/dL and the patient has a history of ischemic heart disease.

Leukocytosis is often noted and is usually due to response to trauma rather than infection.

A platelet count of >100,000/L is safe for neuraxial blockade.

Blood grouping and cross-match: Revision surgery or periprosthetic fractures incur greater blood loss and hence adequate blood units must be cross-matched. Cell salvage should be considered if facilities are available. If massive blood transfusion is anticipated then blood components may be required.

Serum electrolytes: Hyperkalemia may indicate rhabdomyolysis. Hypokalemia with hypovolemia may precipitate atrial fibrillation. Hyponatremia is a common finding in elderly and may result from treatment with thiazide diuretics.

Blood sugar levels: Hyperglycemia should not be a reason to postpone surgery unless the patient is severely dehydrated and/or ketotic.

Electrocardiography: Should be done in all elderly patients.

Chest X-ray: Information in chest X-ray may provide useful with new onset of heart failure and pneumonia or chest injuries.

2D echocardiography: In an elective setting, echocardiography has a role but in the emergent setting; it may lead to delay in surgery. If patient has good effort tolerance, patient may be taken up for surgery in emergency setting. Any known cardiac murmur or a new onset murmur warrants a 2D echocardiography. Departmental guidelines for expediting echocardiography in hip fracture patients with cardiac murmur should be made in liaison with the cardiology department.

Q. How will you optimize the patient preoperatively?

Ans. As soon as the patient arrives in the emergency department simultaneous assessment and optimization should be initiated. Monitoring should be instituted with pulse-oximetry, respiratory rate, ECG, noninvasive blood pressure (NIBP), temperature, static, and dynamic pain scores. This should be followed by:

- **Preoperative traction:** Routine use of preoperative skin and skeletal traction is not recommended.
- **Oxygen therapy:** Pulse oximetry is mandatory from the time of emergency admission to 48 hours after surgery, and oxygen administered as necessary. There is some evidence of supporting routine use of oxygen therapy for the first 72 hours after surgery. Hypoxemia is common after proximal femur fractures and may be due to the pathophysiology of the fracture, or the effect of bed rest, or decreased reserve in the elderly, or coexisting diseases. Any preexisting pulmonary compromise may get magnified after patient suffers from a fracture (e.g., fat embolism or atelectasis).
- **Prevention of pressure sores:** A pressure-relieving mattress should be used for all patients. Patients at very

high-risk of pressure sores should ideally be nursed on a large-cell, alternating-pressure air mattress or similar device.

- **Thromboprophylaxis:** Hip fracture patients should receive low molecular weight heparin (LMWH). Mechanical devices should be used for patients in whom anticoagulants and antiplatelet agents are contraindicated. International normalized ratio (INR) should be <2 for surgery and <1.5 for neuroaxial anesthesia. Patients having warfarin should have vitamin K or prothrombin complex concentrates to reverse the effect (see below).
- **Pressure gradient stockings:** Patients should be wearing pressure gradient stockings as soon as possible after admission.

Q. This patient is on aspirin 75 mg once daily. Many other elderly patients will be on dual antiplatelet agents (aspirin and clopidogrel), having undergone percutaneous coronary intervention and stent placement. How will you manage these patients?

Ans. The Association of Anaesthetists of Great Britain and Ireland (AAGBI) suggests not to stop clopidogrel, especially in patients with drug-eluting stents. An individual approach taking into account the risks and benefits of stopping clopidogrel is needed for each individual patient. Low-dose aspirin may be continued perioperatively. In this patient, aspirin should be continued throughout the perioperative period. In patients who have previously undergone percutaneous coronary intervention (PCI) with stent placement and are on dual antiplatelet agents, elective noncardiac surgery should be delayed, if possible. The details about the management of patients on dual antiplatelet agents can be read in Chapter 2, discussing management of patients with ischemic heart disease.

Q. Elderly patients are often on oral anticoagulants such as warfarin or others, such as novel oral anticoagulants (NOACs—such as apixaban, dabigatran, rivaroxaban, and edoxaban) for atrial fibrillation. How will you manage these patients?

Ans. Intravenous vitamin K can be used to reverse warfarin, but has a slow effect and will take up to 16–24 hours for reversal. Warfarin can be urgently reversed using fresh frozen plasma (FFP), but this carries a risk of volume overload, transfusion-related acute lung injury (TRALI), and transfusion reactions. FFP is rarely able to normalize the INR completely. National Institute for Health and Care Excellence (NICE) guideline [NG39], 2016—Major trauma: assessment and initial management suggest avoiding plasma for reversal of major bleeding in trauma patients.

Prothrombin complex concentrate (PCC) 4-factor, which contains coagulation factors II, VII, IX and X, proteins

C and S, has been extensively studied for reversal of vitamin K antagonists, now available in India (Octaplex), is an attractive alternative. The dose suggested is 25–50 units/kg and it is quite costly.

For reversal of NOACs, 15 mg/kg or 1 g bolus dose of tranexamic acid may be given. If this does not help, patients may be offered hemodialysis. Idarucizumab (a humanized Fab fragment, has a binding affinity more than 350 times that of dabigatran to thrombin) can be used, if available. Andexanet, which is a recombinant engineered version of human factor Xa, can be used as an antidote to any of the NOACs, and is quite effective. Another agent that is currently in the pipeline, Ciraparantag, will be effective against all currently available NOACs.

Q. How will you manage a patient on antidiabetic medications?

Ans. This patient is on glimepiride, a secretagogue from second-generation sulfonylurea class of drugs. It works by stimulating the release of insulin from the pancreatic beta cells [by blocking ATP-sensitive potassium channels (K_{ATP} channels) and causing depolarization of the beta cells] and also possibly by increasing the insulin-mediated peripheral glucose uptake. It can cause hypoglycemia in a fasting patient and should be omitted on the day of surgery. For further details on the management of patients with diabetes mellitus, please see Chapter 34.

Q. What is the ideal time of surgery?

Ans. Surgery should be performed within 48 hours of hospital admission after hip fracture. Delaying surgery beyond 48 hours from admission is associated with prolonged hospital stay, increased morbidity (pressure sores, pneumonia, thromboembolic complications) and increased mortality.

Q. What is the anesthetic technique of choice?

Ans. Anesthetists tend to use a technique with which they are familiar, roughly half-administering regional anesthesia and the remainder general anesthesia. It is important to remember the limited reserves and comorbidities of the elderly patients.

According to a 2016 Cochrane database review, there was no difference between the two anesthetic techniques, except for deep venous thrombosis (DVT), in the absence of potent thromboprophylaxis and reduced operative hypotension (if a unilateral or incremental spinal anesthesia is used).

Q. What are the options available when you plan regional anesthesia? Describe advantages and disadvantages.

Ans.

- *Subarachnoid anesthesia:* This is quick to administer and hence beneficial in the patient with pain. It is commonly

given with low doses of intrathecal bupivacaine (<10 mg) to decrease the incidence of associated hypotension. Also, to reduce the risk of hypotension; unilateral subarachnoid block is preferred (using lower volume of hyperbaric solution with the affected side down). When it is difficult to position the patient with the affected side down, it may be useful to use an isobaric solution (preservative free) to achieve sensory-motor block. Addition of opioids also helps in prolonging postoperative analgesia. Sedation should be used with caution in the elderly and supplemental oxygen should be provided.

- *Epidural anesthesia:* It might be difficult to position the patient for inserting the epidural catheter due to the acute pain. The advantage of this technique is that the duration of anesthesia can be prolonged. Also, local anesthetic dose can be administered in increments and hence may be better tolerated in the hypovolemic and morbid elderly patients.
- *Combined spinal epidural anesthesia:* This combination is of help as there is faster onset of action due to the subarachnoid block. The duration of block can be extended by using the epidural catheter. The downside is the time required for the procedure. This can be shortened with expertise.

Q. How would you administer general anesthesia?

Ans. Graduated doses of anesthetics need to be given to maintain hemodynamics. Inhalational induction is better tolerated and advocated by many. Intraoperative higher inspired oxygen concentration may need to be administered as hypoxemia is common.

Q. Describe perioperative analgesia.

Ans. Surgery is the best analgesic for fracture femur. Preoperative analgesia is required so that the patient is comfortable and cooperative for positioning for anesthesia. Blockade of the femoral, obturator and lateral cutaneous nerve of thigh (3-in-1 femoral nerve block/Psoas compartment block) is an effective method of providing analgesia to patients with hip fracture in the emergency department and is useful for reducing postoperative pain.

Femoral nerve block or the fascia iliaca compartment block (FICB) may not block all the three nerves but causes adequate analgesia for appropriate positioning for the neuraxial blockade. This block can be achieved by the anterior approach under ultrasound guidance as well. Also, a catheter can be placed for continuous local anesthetic infusions in the postoperative period.

Q. What intraoperative monitoring will you use?

Ans. Basic monitoring includes the continual presence of the anesthetist, pulse oximetry, capnography, ECG and

noninvasive blood pressure monitoring. Core temperature monitoring should be used routinely; as elderly patients are at the risk of hypothermia, which is increased if blood transfusions are given. It is also recommended to have point-of-care hemoglobin analyzers to assess anemia and guide blood transfusion. As these patients have high incidence of significant comorbidities; further monitoring may be established, which may include:

Invasive arterial blood pressure monitoring: In patients with limited left ventricular function or valvular heart disease and also in patients with massive blood loss with hypotension. It can also be used for multiple sample collection. Use of pulse pressure variation (PPV) may help in intraoperative fluid management in a mechanically ventilated patient.

Central venous pressure (CVP) monitoring: It may be indicated in patients with limited left ventricular function or undergoing revision of periprosthetic fracture surgery.

Cardiac output monitoring: Transesophageal Doppler may be used to guide fluid therapy under anesthesia. Transthoracic Doppler probes are becoming available for use in sedated or awake patients. Dilution techniques [e.g., lithium dilution cardiac output (LiDCO)] are increasingly accurate, and may be used in conjunction with invasive blood pressure monitoring. Use of stroke volume variation (FloTrac™/Vigileo™ and the PiCCOplus™ system) for fluid responsiveness is associated with better outcomes.

Bispectral index (BIS): May be considered to optimize the depth of anesthesia and avoid potential cardiovascular depression.

Cerebral oxygen saturation: The homeostatic regulation of cerebral blood flow is poor in older patients and depressed further by anesthesia. Thus, monitoring cerebral oxygen saturation may help in reduction of postoperative cognitive dysfunction.

Q. What are the causes of intraoperative hypotension?

Ans.

1. **Hemorrhage:** Patients with fracture femur can lose considerable amount of blood even before reaching the hospital. Moreover, blood loss also occurs during surgery from the raw bone and muscles which may be more than from the identifiable bleeders. Thus, one should be careful while assessing blood loss during the surgery. Special attention should be given to the concealed blood loss in the drapes and the peri-incisional area. It is prudent to do point-of-care hemoglobin estimation to estimate blood loss and assess the requirement of blood transfusion.
2. **Fat embolism syndrome (FES):** Various diagnostic criteria for FES are available (see later). Fat embolism as a subclinical event occurs in majority of the cases of long

bone fracture. The gravity of situation depends on the amount of fat embolized, concurrent medical disease, and aggravating factors. During the intramedullary reaming of femur, there is alteration of the medullary blood supply. As the intramedullary pressure rises, there is reversal of the direction of blood flow (becomes centrifugal instead of centripetal). This leads to intravasation of bone marrow or fat into the vascular system. Arterial hypoxemia may be the only clinical manifestation of subclinical FES whereas hypoxemia, tachycardia, hypotension and fever are early signs preceding the development of FES. Chest radiograph findings are inconsistent; ECG may show ST-segment changes consistent with ischemia. Signs of right heart strain may be present. Once the syndrome has developed, it is associated with high mortality (10–15%). Venting medullary cavity and induced hypotension can reduce the amount of embolism. Laboratory findings of the FES are—a rapid fall in hematocrit, thrombocytopenia, elevation of fibrinogen degradation products, prolonged prothrombin time (PT) and partial thromboplastin time (PTT), and raised serum lipids—free fatty acid and triglycerides. *Occasionally, a patient may present 72 hours after long bone fracture with features of fat embolism and a classical triad of dyspnea, confusion and petechiae.* Criteria for diagnosis for FES are as follows:

Gurd's criteria: For the diagnosis of fat embolism syndrome, at least one major and four minor criteria must be present.

Major criteria:

- Axillary or subconjunctival petechiae
- Hypoxemia $\text{PaO}_2 < 60$ mm Hg; $\text{FiO}_2 = 0.4$
- Central nervous system depression disproportionate to hypoxemia
- Pulmonary edema.

Minor criteria:

- Tachycardia < 110 bpm
- Pyrexia $< 38.5^\circ\text{C}$
- Emboli present in the retina on fundoscopy
- Fat present in urine
- A sudden inexplicable drop in hematocrit or platelet values
- Increasing erythrocyte sedimentation rate (ESR)
- Fat globules present in the sputum.

Lindeque's criteria:

- Sustained $\text{PaO}_2 < 8$ kPa
- Sustained PCO_2 of > 7.3 kPa or a $\text{pH} < 7.3$
- Sustained respiratory rate > 35 breaths/min, despite sedation
- Increased work of breathing: dyspnea, accessory muscle use, tachycardia, and anxiety.

Schonfeld's criteria: Score of >5 is required for diagnosis (Table 1).

TABLE 1: Schonfeld's criteria for the diagnosis of fat embolism syndrome.

Criteria	Score
Petechiae	5
Chest X-ray changes (diffuse alveolar infiltrates)	4
Hypoxemia ($\text{PaO}_2 < 9.3 \text{ kPa}$)	3
Fever ($>38^\circ\text{C}$)	1
Tachycardia ($>120 \text{ beats/min}$)	1
Tachypnea ($>30 \text{ bpm}$)	1

3. Bone cement implantation syndrome (BCIS):

Cardiovascular reactions to acrylic bone cement in patients with fracture neck femur and THR are a common complication. Elderly patients with fractures of the femoral neck constitute a special risk group. Methyl methacrylate (MMA) cement interdigitates within the interstices of cancellous bone, and strongly binds the prosthetic device to the patient's bone. Mixing polymerized MMA (PMMA) powder with liquid MMA monomer causes polymerization and cross-linking of polymer chains. This exothermic reaction leads to cement hardening and expansion against prosthetic components. The resultant intramedullary hypertension can cause embolization of fat, bone marrow, cement, and air into the femoral venous channels. The residual monomer can also cause vasodilatation and a decrease in systemic vascular resistance, thought to be the cause for the transient hypotension often seen with cement insertion. This bone cement implantation is associated with histamine release. In the elderly patients with pre-existing cardiac diseases and/or hypovolemia, even moderate histamine release can cause serious cardiovascular complications. Also, the release of tissue thromboplastin may trigger platelet aggregation, microthrombus formation in the lungs, and cardiovascular instability as a result of circulation of vasoactive substances. The endogenous cannabinoids, anandamide (ANA) and 2-arachidonoylglycerol (2-AG) are also reported to be strong vasodilators and play a role in the hypotension associated with hemorrhagic and septic shock. The clinical manifestations of this syndrome are hypoxemia (increased pulmonary shunt), hypotension, dysrhythmias (including heart block and sinus arrest), pulmonary hypertension, and decreased cardiac output. Occasionally, it can be fatal.

Treatment: BCIS may be reversible with prompt basic life support, combined with treatment to maintain both coronary perfusion pressure and right heart function. An anesthesiologist ordinarily manages this intervention of

supporting the cardiovascular system, treating right heart failure, administering 100% oxygen, and maintaining aggressive volume support. When CVP is high, fluid infusion should be stopped. Vasopressors, such as phenylephrine or norepinephrine, can be titrated to restore adequate aortic perfusion. To improve contractility and ventricular function, inotropes such as dobutamine can be started provided there is adequate right ventricular perfusion pressure to meet the increased oxygen demand caused by these agents. If this syndrome does not result in sudden cardiac death, it may persist for several hours. BCIS is a time-limited process. BCIS is generally reversible even in elderly patients, if their hemodynamic stability is maintained by supportive therapy.

Strategies to minimize the effects of this complication include:

- Good anesthesia technique:
 - Increase inspired oxygen concentration prior to cementing
 - Maintaining normovolemia, monitor blood loss carefully.
- Good surgical technique:
 - Good hemostasis before cementing
 - Good intramedullary lavage
 - Minimizing length of prosthesis
 - Minimizing the pressure applied during insertion
 - Venting the distal femur to relieve intramedullary pressure
 - Use uncemented femoral component.

Q. Discuss DVT and thromboembolism.

Ans. Venogram and ventilation/perfusion studies have shown a prevalence of 37% for DVT and 6% for pulmonary embolism (PE), although clinical symptoms are only seen in 1–3% of DVTs and 0.5–3% of PEs in patients with hip fracture.

Factors responsible for this are:

- Preoperative as well as postoperative immobilization—more so, if it is a prolonged one
- Decreased blood flow to the limb, e.g., kinking of femoral vessels
- Postoperative hypercoagulable state.

Deep venous thrombosis prophylaxis must be given to all elderly patients undergoing orthopedic or pelvic operations who are likely to be confined to bed for more than a day, especially those having malignancy as they are at high risk of DVT and PE. Either standard unfractionated heparin (UFH) (5,000 units SC BD or TDS) or LMWH should be given. Respiratory distress, unexplained bronchospasm, hypotension, or hypoxia occurring during or a few days after surgery should raise the suspicion of PE. Any atypical pneumonia not responding to antibiotics, tachycardia or dry cough with the classic triad of dyspnea, hemoptysis and pleural pain should warn an anesthetist

of a possible PE. Arterial blood gas (ABG) may show hypoxia and hypocapnea. The ECG may show a $S_1Q_3T_3$ pattern (very rarely), and the X-ray chest may show a cut-off of the pulmonary vasculature, pulmonary infarction or a wedge-shaped opacity. However, both ECG and X-ray are commonly normal in most patients with PE. Intravenous heparin should be started if PE is suspected, while investigations such as V/Q scan, echocardiography (to rule out right-sided dysfunction), spiral CT scan or pulmonary angiography are carried out. Treatment consists of heparin infusion. Hemodynamically unstable patients may be considered for thrombolysis up to 2 weeks after the onset of PE, but you may not be able to give it in immediate postoperative period. In severe cases, in appropriately equipped centers, surgical embolectomy can be performed.

Q. What other precautions should be taken intraoperatively?

Ans. Thromboprophylaxis: LMWH or UFH or fondaparinux are commonly prescribed to these patients. The timing of administration of heparin should be adjusted so that it does not interfere with neuraxial blocks. Thromboembolism stockings or intermittent compression devices should be used in the perioperative period as well. Early surgery, regional anesthesia and mobilization reduce the risk of DVT.

Antibiotics: Appropriate antibiotic prophylaxis according to the local hospital protocols should be used.

Pressure care: Elderly are prone to damage due to pressure. They should be positioned for surgery carefully and pressure points should be padded adequately to avoid pressure sores and neuropraxia. The skin is thin, fragile and liable to be damaged by minimal trauma like removal of dressings, ECG electrodes, diathermy plates, and moving of the patient; hence, utmost care should be taken.

Thermoregulation: Elderly patients are prone to hypothermia in the perioperative period especially in the intraoperative period. Adequate warming strategies should be employed to maintain temperature. Fluid warmers and convective warming blankets should be routinely used.

Intravenous fluids: These patients are at high risk of hypovolemia due to blood loss. Active fluid management starting in the preoperative period is essential to maintain blood pressure and urine output. Ringer lactate solution is usually used, however, saline may be used to maintain preload. Plasma expanders (gelatins, starch, etc.) may be required when blood products are still to be given. CVP often fails in providing information regarding cardiac preload and it does not predict fluid responsiveness. In this situation, assessing fluid responsiveness with stroke volume variation (SVV) or PPV is helpful.

Q. What care should be taken in the postoperative period?

Ans. As most of the patients are elderly and with multiple comorbidities, they are at relatively high risk of complications and hence may require a prolonged period of monitoring in the postoperative period.

Analgesia: Analgesia requirements vary from patient to patient especially during remobilization. Single shot peripheral nerve blockade does not last long. In addition, 1 g acetaminophen IV or oral (as applicable) administration should continue. Continuous infusion of local anesthetics helps in relieving pain during the postoperative period. NSAIDs and opioids, intravenous or oral, should be used with caution. Pain scores should be regularly charted to evaluate the treatment.

Oxygen: Oximetry assessment should guide oxygen therapy. Some studies suggest oxygen therapy for 72 hours in the postoperative phase. Mobilization helps in improving oxygenation and respiratory function.

Fluid balance: Early oral fluid intake should be encouraged as soon as possible. Urinary catheters should be removed at the earliest to decrease the risk of urinary tract infection.

Wound care: Avoiding staplers for skin closure is suggested to decrease the rate of infection.

Postoperative cognitive dysfunction/acute confusional state/delirium: This is common and interrupts rehabilitation of these patients. Prevention is the best treatment of postoperative cognitive dysfunction. Optimizing analgesia, nutrition, hydration, electrolyte balance, appropriate medication, bowel habit and mobilization, in conjunction with identifying and treating complications such as chest infection, silent myocardial ischemia and urinary tract infection help in preventing delirium. Drugs such as haloperidol or lorazepam may be used to tide over the crisis cautiously.

Nutrition: Most patients are malnourished. The calorie requirement should be calculated for individual patients and supplemented accordingly.

Rehabilitation: This starts post surgery and continues even after discharge from the hospital. It should be aimed at getting the patients to their preinjury status. Multidisciplinary approach is required for adequate rehabilitation and recovery, and should include physiotherapists, occupational therapists, social workers, nursing staff and their own relatives.

Q. Discuss postoperative thromboprophylaxis.

Ans. Unless contraindicated, all postoperative patients should be on thromboprophylaxis for prevention of DVT and

subsequent PE. In patients undergoing major orthopedic surgeries, the incidence of DVT ranges from 40% to 60%. Virchow's triad described (1884) the pathophysiologic processes involved in development of DVT; this included the following: vascular endothelial damage, stasis of blood flow, and hypercoagulability of blood. The orthopedic surgeries have all these elements present:

- Intraoperative tourniquet use, immobilization and bed rest, all causing stasis of venous blood.
- Surgical intervention in the limb and handling of vessel leading to possible vascular endothelial injury.
- Trauma, which increases thromboplastin or thrombokinase (also called coagulation factor III, thromboplastin or tissue factor) levels, and PMMA bone cement which increases hypercoagulability.

All these make the elderly patients particularly susceptible to DVT and/or PE. These patients should be, therefore, on a combination of LMWH or UFH (if renal failure is present) and sequential calf compression device.

Q. How will you decide when to remove the epidural catheter?

Ans. See Chapter 52 on *Regional Anaesthesia Instruments* for detailed discussion on the timing of removal of the epidural catheter.

To manage patients on anticoagulation, basic pharmacokinetic rules to observe include the following:

- Not performing catheter removal until at least 2-T_{1/2} (T_{1/2} depending on renal and hepatic function) after last anticoagulant administration for optimal risk/benefit ratio [25% pharmacodynamic efficacy or being more conservative with 5-T_{1/2} (3.125% anticoagulant in circulation) in high-risk patients or from new anticoagulant with limited clinical experience].
- Following catheter removal/neuraxial and deep needle puncture, next anticoagulant administration should be based on the time required to reach maximum activity.
- Clinical vigilance during initial hours or days.

For UFH: Remove catheter 4–6 hours after the dose.

For LMWH:

- *For single daily dosing:* Catheter should be removed 10–12 hours after the last dose of LMWH. Subsequent LMWH dosing should occur at a minimum of 2 hours after catheter removal.
- *For twice daily dosing:* Indwelling catheter should be removed before the initiation of LMWH thromboprophylaxis.

For warfarin: Neuraxial catheter should be removed when INR is less than 1.5.

Fondaparinux: Catheters to be removed 36 hours after the last dose, and subsequent dosing to be delayed for 12 hours after catheter removal.

European Society of Regional Anesthesia (ESRA) recommended time intervals before and after neuraxial puncture or catheter removal (when renal function is normal) are listed in **Table 2**.

TABLE 2: European Society of Regional Anesthesia (ESRA) recommended time intervals before and after neuraxial puncture or catheter removal.

	Time before puncture/catheter manipulation or removal	Time after puncture/catheter manipulation or removal
UFHs (for prophylaxis, ≤15,000 IU/d)	4–6 h	1 h
UFHs (for treatment)	IV 4–6 h SC 8–12 h	1 h 1 h
LMWHs (for prophylaxis)	12 h	4 h
LMWHs (for treatment)	24 h	4 h
Fondaparinux (for prophylaxis, 2.5 mg/d)	36–42 h	6–12 h
Rivaroxaban (for prophylaxis, 10 mg daily)	22–26 h	4–6 h
Apixaban (for prophylaxis, 2.5 mg BID)	26–30 h	4–6 h
Dabigatran (for prophylaxis, 150–220 mg)	Contraindicated according to the manufacturer	6 h
Coumarins	INR ≤1.4	After catheter removal
Hirudins (desirudin)	8–10 h	2–4 h
Argatroban	4 h	2 h
Acetylsalicylic acid	None	None
Clopidogrel	7 d	After catheter removal
Ticlopidine	10 d	After catheter removal
Prasugrel	7–10 d	6 h after catheter removal
Ticagrelor	5 d	6 h after catheter removal
Cilostazol	42 h	5 h after catheter removal
NSAIDs	None	None

(INR: international normalized ratio; LMWH: low molecular weight heparin; NSAIDs: nonsteroidal anti-inflammatory drugs; UFH: unfractionated heparin).

Q. What preventive measures may be advised to the patients?

Ans.

- *Osteoporosis treatment:* Vitamin D and calcium supplementation are recommended for osteoporosis in the elderly. Vitamin D supplementation, injected or given orally, suppresses parathyroid hormone,

increases bone mineral density. It is shown that older people in institutions may sustain fewer hip and other nonvertebral fractures, if given vitamin D with calcium supplements.

- An annual IV infusion of 4 mg zoledronic acid is associated with a reduction in rate of new clinical vertebral and nonvertebral fractures and may improve survival after a low-trauma hip fracture. Oral alendronate and oral risedronate are associated with reductions in rates of vertebral and nonvertebral fractures.
- *Hip protectors:* They may reduce the risk of hip fracture in institutionalized patients, but not in community-dwelling older people. Patient acceptance of hip protectors and adherence to their use remain poor due to discomfort and practicality.

Q. What ethical issues should be considered?

Ans. Ethical issues arising while treating elderly patients with hip fracture are: Consent—elderly patients may have mild-to-severe cognitive impairment. Also, the ability to assimilate information and communicate decisions may be impaired by poor vision, hearing or speech. Thus, if the patient is not in the capacity to consent for surgery, next of kin or the legal guardian should be consulted about treatment options and decision made.

SUGGESTED READING

1. Agu O, Hamilton G, Baker D. Graduated compression stockings in the prevention of venous thromboembolism. *Br J Surg.* 1999;86:992-1004.
2. Amaragiri SV, Lees TA. Elastic compression stockings for prevention of deep vein thrombosis. *Cochrane Database Syst Rev.* 2000;(3):CD001484.
3. Association of Anaesthetists of Great Britain and Ireland (AAGBI). (2007). Recommendations for Standards of Monitoring during Anaesthesia and Recovery, 4th edition. [online] Available from <http://www.aagbi.org/publications/guidelines/docs/standardsofmonitoring07.pdf>. [Last accessed August, 2012].
4. Association of Anaesthetists of Great Britain and Ireland, Griffiths R, Alper J, Beckingsale A, Goldhill D, Heyburn G, et al. Membership of the Working Party. Management of proximal femoral fractures 2011: Association of Anaesthetists of Great Britain and Ireland. *Anaesthesia.* 2012;67: 85-98.
5. Balakrishnan K. Care of the patient with fracture neck of femur for non-emergency surgery. *Sri Lankan J Anaesthesiol.* 2010;18:46-52.
6. Donaldson AJ, Thomson HE, Harper NJ, Kenny NW. Bone cement implantation syndrome. *Br J Anaesth.* 2009;102: 12-22.
7. Fletcher AK, Rigby AS, Heyes FL. Three-in-one femoral nerve block as analgesia for fractured neck of femur in the emergency department: a randomized, controlled trial. *Ann Emerg Med.* 2003;41:227-33.
8. Foss NB, Kristensen MT, Jensen PS, Palm H, Krashennikov M, Kehlet H. The effects of liberal versus restrictive transfusion thresholds on ambulation after hip fracture surgery. *Transfusion.* 2009;49:227-34.
9. Guay J, Parker MJ, Gajendragadkar PR, Kopp S. Anaesthesia for hip fracture surgery in adults. *Cochrane Database Syst Rev.* 2016;2:CD000521.
10. Handoll HHG, Farrar MJ, McBirnie J, Tytherleigh-Strong G, Milne AA, Gillespie WJ. Heparin, low molecular weight heparin and physical methods for preventing deep vein thrombosis and pulmonary embolism following surgery for hip fractures. *Cochrane Database Syst Rev.* 2002;(4):CD000305.
11. Khan SK, Kalra S, Khanna A, Thiruvengada MM, Parker MJ. Timing of surgery for hip fractures: a systematic review of 52 published studies involving 291,413 patients. *Injury.* 2009;40:692-7.
12. Li J, Halaszynskin T. Neuraxial and peripheral nerve blocks in patients taking anticoagulant or thromboprophylactic drugs: challenges and solutions. *Local Reg Anesth.* 2015;8:21-32.
13. Luger TJ, Kammerlander C, Gosch M, Luger MF, Kammerlander-Knauer U, Roth T, et al. Neuroaxial versus general anaesthesia in geriatric patients for hip fracture surgery: does it matter. *Osteoporos Int.* 2010;21(Suppl 4):S555-72.
14. Mak JCS, Cameron ID, March LM; National Health and Medical Research Council. Evidence-based guidelines for the management of hip fractures in older persons: an update. *Med J Aust.* 2010;192:37-41.
15. Minville V, Asehnoun K, Salau S, Bourdet B, Tissot B, Lubrano V, et al. The effects of spinal anesthesia on cerebral blood flow in the very elderly. *Anesth Analg.* 2009;108(4):1291-4.
16. Parker MJ, Griffiths R, Appadu BN. Nerve blocks (subcostal, lateral cutaneous, femoral, triple, psoas) for hip fractures. *Cochrane Database Syst Rev.* 2002;(1):CD001159.
17. Parker MJ, Handoll HHG, Griffiths R. Anaesthesia for hip fracture surgery in adults. *Cochrane Database Syst Rev.* 2004;(4):CD000521.
18. Scottish Intercollegiate Guidelines Network. (2009). Management of hip fracture in older people. A national clinical guideline. [online] Available from <https://www.sign.ac.uk/assets/sign111.pdf>. [Last accessed March, 2020]
19. Shiga T, Wajima Z, Ohe Y. Is operative delay associated with increased mortality of hip fracture patients? Systematic review, meta-analysis and meta-regression. *Can J Anaesth.* 2008;55(3):146-54.

Cataract

Sumitra Ganesh Bakshi, Debashree Lahiri, Sudivya Sharma

A 75-year-old lady is posted for a cataract surgery with an intraocular lens implant.

Q. How would you evaluate this patient preoperatively?

Ans. This patient is an elderly female and expected to have age-related illness which should be sought for accordingly. This aspect has been dealt with in detail in a separate chapter.

"Anesthetic Considerations in a Geriatric Patient." In short, in elderly patients ask for the following comorbidities:

- **Hypertension:** Duration, medication, effort tolerance, history of ischemic heart disease (IHD), any anticoagulant therapy.
- **Diabetes:** Duration, medications, any end-organ damage or autonomic dysfunction if present.
- **Respiratory problem:** Symptoms, duration, medication, dyspnea on lying down would make the patient uncomfortable under regional anesthesia.
- **Orthopedic problem:** Degenerative spine (cervical), joint pain, back pain, which prevents patient from lying down for a long time.
- **Central nervous system (CNS) and psychiatric illness:** In elderly, there is increased incidence of functional illness and emotional disturbances. Alzheimer disease, titubation (involuntary head movements) could pose a problem during surgery under regional anesthesia.

Also note—if patient has deafness, dentures, language difficulties, dizziness or claustrophobia.

Q. What preoperative investigations you would ask for this procedure?

Ans. Cataract surgery is a relatively low-risk surgery in a high-risk patient. There exists a controversy in the extent of investigations to be done for a patient undergoing this surgery under regional anesthesia or monitored anesthesia care (MAC). As per the 2002 American Society of Anesthesiologists (ASA) preoperative testing advisory no tests are required for minimally invasive surgery, if the patient is clinically fit. Hence, the number of investigation

would depend upon anesthesia technique, and institutional guidelines.

In cases of planned general anesthesia a detailed work up as per associated medical illness would be needed. In pediatric cases, echocardiography to look for cardiac involvement and X-ray neck lateral to look for vertebral anomalies should be performed in addition to a complete hemogram. In general, cataract surgery is a planned case. However, it is of urgent nature when:

- **Intraocular tension (lens-induced glaucoma) is raised:** These patients are generally started on tablet acetazolamide or injection mannitol to reduce intraocular pressure (IOP). Both drugs cause electrolyte imbalance.
- In infants, to prevent suppression amblyopia.

Q. Discuss the perioperative management of anticoagulants and antiplatelets in a patient undergoing cataract surgery?

Ans.

- For patients undergoing cataract surgeries, a detailed history of antiplatelet drugs and their indications is essential. It is advisable to inform patients preoperatively about the risk of orbital bleeding if antiplatelet/anticoagulant agents are continued, as well as the risk of thromboembolic events on discontinuing them.
- To summarize the key recommendations from the American Academy of Ophthalmology and Brazilian Society of Cardiology:
 - Continue warfarin in patients undergoing cataract surgery, if the INR is in the therapeutic range.
 - In case the INR is elevated (3–4), review is essential.
 - For patients on antiplatelet agents for secondary prevention of coronary heart disease, aspirin or clopidogrel should be continued during cataract surgery.

- Patients requiring dual antiplatelet therapy for a defined period of time following angioplasty based on the type of stent inserted. Cataract surgery if possible should be postponed for the duration.
- However, if cataract surgery is urgent the guidelines indicate that double antiplatelet therapy should not be stopped.
- Ophthalmic bleeding events associated with cataract surgery are generally few and usually do not have serious consequences, even if antiplatelet/anticoagulant medications are continued perioperatively.
- Discontinuation of these drugs may lead to potentially serious thromboembolic events increasing the morbidity or mortality.
- However, if they have to be discontinued preoperatively in other ophthalmic surgeries (like vitreous surgeries), close consultation between the surgeon and the physician is mandatory in order to limit the risks associated with their stoppage.
- Topical anesthesia is preferred for patients taking these medications.

Q. What is the difference between preoperative evaluation of this patient as against a pediatric patient coming for cataract surgery?

Ans.

In cases of congenital cataract:

One should note the details of antenatal care (ANC), e.g., history suggestive of maternal rubella. Often, the eye finding is a part of a syndrome. History and investigations to rule out other system involvement is a must.

Common syndromes include:

- **Down syndrome:** To rule out heart defects, thyroid problem, seizures. Difficult airway in presence of macroglossia is common.
- **Galactosemia:** Galactose intolerance affects liver, kidney, brain, and eyes.
- Other syndromes are congenital rubella syndrome, Pierre Robin syndrome, chondrodysplasia syndrome, Lowe syndrome, trisomy 13.

Q. What are the various anesthetic techniques that may be used in these patients?

Ans.

Choice of anesthesia depends upon:

- **Surgical choice:**
 - Need for complete akinesia
 - Duration of surgery
 - Extent of surgery.
- **Patient factors:**
 - Age and patient's cooperation for the procedure
 - Associated medical conditions.

Anesthetic techniques available are:

- Regional
- Retrobulbar block ± Facial block
- Peribulbar block
- Sub-Tenon's block
- Topical anesthesia
- Regional + Monitored anesthesia care
- General anesthesia ± Regional.

Q. What are the advantages of local blocks over general anesthesia?

Ans.

- Patients can be safely discharged home on same day
- Produce good akinesia and anesthesia
- Minimal influence on IOP
- Require minimum equipment.

Q. What are the contraindications to blocks?

Ans.

- Procedures lasting significantly more than 90 minutes
- Bleeding or coagulation disorders
- Perforated globe
- Allergy to local anesthetics (LAs)
- Disorientation or mental impairment
- Inability to lie flat.

Q. Describe relevant anatomy with nerve supply of the eyeball?

Ans. Anatomy: Each orbit has the shape of an irregular pyramid with its base at the front of the skull and its axis pointing poster medially toward the apex. The depth of the orbit measured from the rear surface of the eyeball to the apex is about 25 mm (range 12–35 mm). The axial length (AL) of the globe (eyeball) is the distance from the corneal surface to the retina and is often measured preoperatively. An AL of 26 mm or more denotes a large eye, indicating that great caution is necessary as globes longer than this are easier to perforate during regional anesthesia.

Orbital fat: It is divided into central (retrobulbar/intracone) and peripheral (peribulbar/pericone) compartments by the cone of the recti muscles. The central space contains the optic, oculomotor, abducent, and nasociliary nerves. The peripheral space contains the trochlear, lacrimal, frontal, and infraorbital nerves. All the motor and sensory nerves can be blocked by an injection into the orbital fat (**Table 1**).

Nerve supply to the eyes: The motor nerve supply to the extraocular muscles is easy to remember using the mnemonic LR₆ (SO₄)₃—lateral rectus by the 6th (abducent) cranial nerve, superior oblique by the 4th (trochlear) and the remainder by branches of the 3rd (oculomotor) nerve.

The parasympathetic supply is from the Edinger-Westphal nucleus accompanying the 3rd nerve to synapse

with the short ciliary nerves in the ciliary ganglion. The sympathetic fibers are from T₁ (the first thoracic sympathetic outflow) and synapse in the superior cervical ganglion before joining the long and short ciliary nerves. **Figure 1** shows the peribulbar and retrobulbar needle position.

TABLE 1: Summary of sensory nerve supply.

Sclera and cornea		• Short ciliary nerves • Long ciliary nerves
Conjunctiva	Superior	• Supraorbital nerve • Supratrochlear nerve • Infratrochlear nerve
	Inferior	Infraorbital nerve
	Lateral	Lacrimal nerve (with contribution from zygomaticofacial nerve)
	Circumcorneal	Long ciliary nerves
Periorbital skin		• Supraorbital • Supratrochlear • Infraorbital • Lacrimal

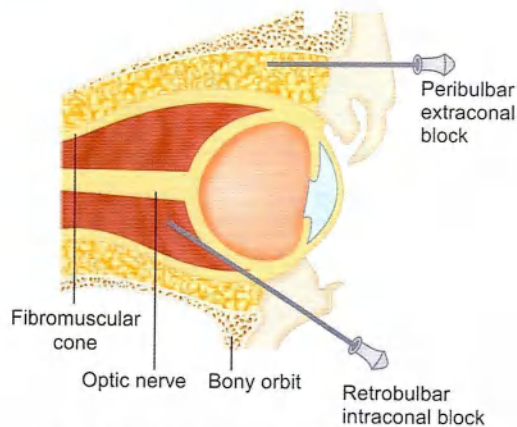


Fig. 1: Peribulbar and retrobulbar needle position.

Q. Discuss peribulbar block (pericone).

Ans. Advantages: Less incidence of optic nerve damage. The incidence of retrobulbar hemorrhage and perforation is the same. Facial block is not needed.

Disadvantages: Since the LA is placed outside the muscle cone the concentration around the optic nerve may not be sufficient to abolish vision completely. Some light perception will therefore remain; however, the patient is not able to see the operation. Periorbital ecchymosis is common. Larger volumes of LA needed.

Procedure: The block is administered by two injections, first through upper lid (at junction of medial one-third and lateral two-thirds) and second through the lower lid (at junction of lateral one-third and medial two-thirds). After orbital injection Honan pressure cuff ball can be applied on eyeball for sustained pressure. Assessment of the block

is usually judged after an interval of 10 minutes. The signs of a successful block are:

- Ptosis (drooping of the upper lid with inability to open the eyes)
- Either no eye movement or minimal movement in any direction (akinesia)
- Inability to fully close the eye once open.

Q. Compare retrobulbar and peribulbar block with regards to technique, advantage, disadvantage, and associated complications.

Ans.

Retrobulbar Block (Intracone)

The LA is delivered within the muscle cone itself, behind the globe. It is aimed at blocking the ciliary ganglion, ciliary nerves, cranial nerves II, III, VI. Cranial nerve IV is not affected since it lies outside the muscle cone (**Fig. 1**).

Procedure: A 24-gauge, 3.5-cm long needle is used. Entry: Transcutaneously at junction of middle and lateral third of lower orbital margin. Once you feel the first pop through the orbital septum (around 15 mm), angle 45° medially and 45° superiorly toward the apex of the orbit until the second pop through the muscle cone is felt.

Facial Block

It is necessary to block the facial nerve which supplies orbicularis oculi muscle, so that patient cannot squeeze the eyelids during surgery. It is needed in most cases with retrobulbar block. A variety of techniques have been described for this. These are (**Fig. 2**):

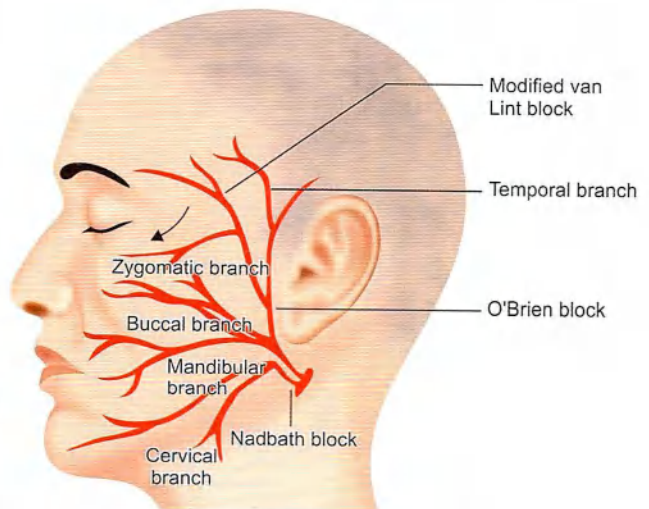


Fig. 2: Major approaches to blocking the facial nerve.

- **van Lint method:** Nerve block close to outer canthus, a wheal is raised 1 cm below and behind the lateral canthus. The needle is passed through the wheal

upward toward the temporal fossa and drug injected while withdrawing, the needle is turned medially and downward toward the infraorbital foramen and LA injected. Lastly LA is injected along the lower margin of zygoma.

- **O'Brien method:** Paralysis of VII nerve, anterior to the condyloid process of the mandible. The needle is inserted directly to hit the bone and LA is injected as the needle is withdrawn.
- **Nadbath block:** The facial nerve is blocked at the stylomastoid foramen.

Q. What are the complications of the retrobulbar block?

Ans. Retrobulbar hemorrhage: It is the most common complication seen and is due to inadvertent puncture of vessels within the retrobulbar space. Signs are proptosis and a palpable increase in IOP. Subconjunctival blood and eyelid ecchymosis may be seen as the hemorrhage extends anteriorly. Retrobulbar hemorrhage can lead to other complications such as central retinal artery occlusion and stimulation of the oculocardiac reflex (OCR). It is best to postpone surgery for 2–4 days if this complication occurs.

Bradycardia: Due to OCR, bradycardia can occur several hours later in the event of an expanding hemorrhage. Intravenous atropine is the treatment of choice.

Central retinal artery occlusion: It can result from retrobulbar hemorrhage or injection of LA into optic sheath and may result in total loss of vision if not treated. A deep lateral canthotomy or an anterior chamber paracentesis to decompress the orbit may be needed.

Puncture of the posterior globe: More likely in patients with a severely myopic eye ("long eye") and in patients with staphyloma. Repeated anesthetic injections using long, sharp needles increases the risk of globe penetration. Perforation usually occurs in inferolateral quadrant. Retinal detachment and vitreous hemorrhage are common sequelae with potential for visual loss. Accidental forceful LA injection into the globe is more hazardous and can cause rupture of globe. Pain at time of block, hypotonia, and sudden loss of vision should alert the anesthesiologist.

Others:

- **Shivering:** Because of absorption of LA along optic nerve sheath into CNS
- Penetration of the optic nerve
- **Inadvertent brainstem anesthesia:** Accidental injection into the cerebrospinal fluid (CSF) due to perforation of the meningeal sheaths that surround the optic nerve
- Injection into muscle leading to muscle dysfunction
- Postoperative ptosis (13%)
- Allergic reactions.

Q. What are the precautions that should be taken to prevent globe perforation?

Ans.

- Excise caution in myopics and patient with staphyloma
- Repeated injections to be avoided
- In such cases peribulbar block is to be preferred
- **Wiggle test:** After retrobulbar needle is in orbit, before any anesthetic is injected the needle is moved from side to side. Any rotation of eye suggests that either the sclera, optic nerve, or extraocular muscle has been penetrated. This test will prevent forceful injection of LA drug into vital areas, though it does not prevent globe perforation in the true sense.

Q. What is a sub-Tenon's block?

Ans. Sub-Tenon or a parabulbar block is block given in the sub-Tenon's capsule or space. A conjunctival incision 2–3 mm in size is made halfway between the inferior limbus and fornix to open into sub-Tenon's space. A blunt cannula is then used to inject anesthetic into posterior sub-Tenon space.

Advantages: Lesser volume is needed. There are less chances of vascular and optic nerve injury.

Q. What do we understand by intracameral injection of local anesthetics?

Ans. Preservative-free anesthetic (usually 1% lidocaine) is injected into the anterior chamber at the beginning of the surgery through one of the corneal incisions required for phacoemulsification. It provides sensory block of the iris and the ciliary body and thereby relieves discomfort experienced during IOL placement. Intracameral injections are common adjust to topical analgesia during phacoemulsification.

However, retinal toxicity can occur because of posterior diffusion of local anesthesia to the retina, and a temporary loss has been reported. Hence preservative-free LA must be used in small amounts. Preservative lidocaine 1% in doses of 0.1–0.5 mL is recommended.

Q. Is topical anesthesia a suitable alternative?

Ans. The terminal branches of cornea and conjunctiva form an intraepithelial plexus. These nerve endings are superficial and vulnerable to the effect of LAs.

Advantages:

- Quick
- Noninvasive
- Well-tolerated by patients
- No complications of other blocks
- Patient has an immediate improvement in vision and eye shield is not needed.

Disadvantages:

- No akinesia
- Patient should be motivated and cooperate

- Only a simple uncomplicated cataract can be included
- Expertise surgical hand needed.

Q. What is oculocardiac reflex (OCR)?

Ans. Oculocardiac reflex, also known as *Aschner-Dagnini reflex*, is bradycardia due to traction on the extraocular muscles, conjunctiva, orbital structures, and pressure on the globe, retrobulbar block, and ocular trauma. The afferent is carried by trigeminal nerve while vagus nerve brings the efferent arm of the reflex. The afferent pathway follows the long and short ciliary nerves to the ciliary ganglion and then to Gasserian ganglion along the ophthalmic division of trigeminal nerve. These afferent pathways terminate in the main trigeminal sensory nucleus in the floor of the fourth ventricle. The efferent impulses start in the muscles of the vagal cardiac depressor nerve, causing negative inotropic and conduction defects. Oculocardiac reflex can also lead to arrhythmia including ventricular tachycardia and rarely asystole. Hypotension and increased arterial carbon dioxide partial pressure significantly increase incidence of bradycardia during surgery. Incidence of OCR is highest in children if no atropine pretreatment is used. In children, administration of atropine 0.02 mg/kg or glycopyrrolate 0.01 mg/kg prior to commencing surgery is indicated. In adults prophylaxis is not usually indicated.

Treatment:

- Removal of stimulus
- Intravenous anticholinergics
- Check and deepen depth of anesthesia
- Lignocaine if ventricular premature contractions (VPCs) are a problem.

Q. What is monitored anesthesia care (MAC)?

Ans. Monitored anesthesia care is a comprehensive physician service that includes all aspects of anesthetic care—preprocedural visit, intraoperative care, and postprocedure anesthetic management. The ASA practice guidelines for MAC have been established. These guidelines frequently are consistent with those for general or regional anesthesia and should be followed accordingly. Although MAC is safe when administered by well-trained anesthesia personnel, ASA closed claims data demonstrate that rare and severe injuries or death can occur during MAC. Clinicians must understand the pharmacokinetic and pharmacodynamic principles of drug administration (e.g., context-sensitive half time, effect-site half-time) so that cognitive errors can be avoided. Proper drug dosing can optimize patient safety and recovery and discharge times. Therapeutic windows for drugs administered during MAC are much smaller than those for general anesthesia. Sedation with propofol is classified as deep sedation by the ASA. When used in doses consistent with MAC, significant changes occur in airway anatomy and cardiopulmonary physiology. These changes

occur even in healthy patients and are amplified in the elderly and in patients with preexisting systemic disease. Propofol should be administered only by clinicians qualified to rescue patients from any level of sedation, including general anesthesia.

Q. Discuss management of a restless patient undergoing cataract surgery under local anesthesia.

Ans.

- Ask surgeon to stop.
- Recheck vitals including saturation.
- Discuss with patient the cause for restlessness and reassure.
- Often patient “feels out of breath” under drapes—in such cases starting of oxygen through mask or cannula is of help. This should be ideally done for all patients. Just lifting the drapes of the face also helps in allaying anxiety and at times preventing rebreathing of CO₂ which is highly probable with the drapes.
- In case patient complains of pain, LA should be repeated in the form of a repeat block or topical drops.
- Intravenous analgesics such as fentanyl and anxiolytics can be considered. One has to titrate these drugs by giving small boluses and observing for the desired effect. Also one should be watchful for airway obstruction and respiratory depression.
- At times if patient does not cooperate, propofol can be used. Oropharyngeal airway or laryngeal mask airway (LMA) should be ready for insertion.
- In a few rare instances, if above interventions do not help, administration of general anesthetic and relaxant with full control of airway with endotracheal tube in situ may be necessary.

Q. What are the systemic effects of ophthalmic drugs?

Ans. Local ocular therapy involves high concentrations of drug molecules in a small volume. Systemic side effects are rare. But caution with its use is required in vulnerable groups including children, elderly, and pregnant ladies (Table 2).

TABLE 2: Systemic effects of ocular drugs.

Medications	Vulnerable population—drug best avoided	Reported systemic adverse effects
<i>Antiglaucoma:</i>		
Brimonidine (alpha-agonists)	Nursing mothers, infants, patients on MAO inhibitors, tricyclic antidepressants	Dry mouth, tachyarrhythmias, headache, taste disturbances, skin rash
Antisymphathotonic agents, e.g., clonidine		Hyperemia, pruritus, foreign body sensation

Contd...

Contd...

Medications	Vulnerable population—drug best avoided	Reported systemic adverse effects
Timolol (non-selective β -blockers)	Asthma, emphysema, COPD, congestive heart failure, bradycardia, sick sinus syndrome, heart block—especially AV blocks (second- and third-degree block)	Urticaria, alopecia, contact dermatitis, or psoriasiform rashes, bronchospasm, headache, insomnia
Acetazolamide (systemic carbon anhydrase inhibitors)	Renal acidosis, Addison's disease, severe renal/liver dysfunction	Paresthesia, metabolic acidosis, fever, urticaria, confusion, sleepiness, depression, gout, renal stones, taste disturbances
Pilocarpine (cholinergics)	None	Headache, vertigo, malaise, asthma symptoms
Latanoprost (prostaglandin analogs)	None	Headache, tachycardia, skin rash, dizziness, exacerbated asthma symptoms, muscle and joint pain, and minor local reactions
Antihistaminics, e.g., ketotifen and olopatadine	None	Headache, nausea, dry mouth, and drowsiness
Antimicrobials, e.g., ofloxacin and tobramycin	None	Contact allergic reactions, eczema, headache, rhinitis, affect taste
Mydriatics, e.g., atropine, tropicamide, and cyclopentolate	None	Dry mouth, fever, tachycardia, gastrointestinal symptoms, confusion and memory disorders, hallucinations and abnormal behavior in children

Contd...

Contd...

Medications	Vulnerable population—drug best avoided	Reported systemic adverse effects
Sympathomimetics, e. g., Phenylephrine	Children and elderly	Contact dermatitis, adverse cardiovascular effects
Nonsteroidal anti-inflammatory drugs, e.g., diclofenac	Acetylsalicylic acid (ASA) sensitivity, asthmatics	Contact dermatitis, itching, and cough
Steroids		Cortisone glaucoma, cataract

(AV: atrioventricular; COPD: chronic obstructive pulmonary disease; MAO: monoamine oxidase).

Adverse Ocular Effects Caused by Systemic Drugs

Certain drugs may have ocular effects too. Chronic glucocorticoid therapy can increase IOP and promote cataract development. Adrenergic or cholinergic drugs have autonomic side-effects, e.g., antipsychotic or antidepressant drugs which may antagonize the cholinergic system and thus increase IOP. Hydroxychloroquine and chloroquine are commonly used for the prevention of malaria and to treat rheumatic and other autoimmune diseases, these drugs are irreversibly toxic to the retina. Ethambutol, antitubercular drug, causes irreversible toxic effect on optic nerve.

SUGGESTED READING

1. Kara-Junior N, Koch CR, de Santhiago MR, Fornari L, Caramelli B. Anticoagulants and antiplatelet drugs during cataract surgery. *Arq Bras Oftalmol.* 2018;81:348-53.
2. Kumar C, Dowd T. Ophthalmic regional anaesthesia. *Curr Opin Anaesthesiol.* 2008;21(5):632-7.
3. Malik A. Efficacy and performance of various local anesthesia modalities for cataract surgery. *J Clin Exp Ophthalmol.* 2013;31:007. doi10.4172/2155-9570.
4. Miller RD. *Miller's Anesthesia*, 6th edition. Philadelphia: Elsevier, Churchill Livingstone; 2005.
5. Sihota R, Tandon R. *Parsons' Disease of the Eye*. Elsevier India, 2014.
6. Vaajanen A, Vapaatalo H. A Single Drop in the Eye—Effects on the Whole Body? *Open Ophthalmol J.* 2017;11:305-14.

Morbid Obesity

Vijaya Prakash Patil, Malini Joshi, Bindiya Salunke

A 53-year-old female patient weighing 105 kg with height of 157 cm presents with history of neck swelling. The swelling has gradually increased over last 5 months and her voice has changed since 4 months. She has developed dysphagia to liquids and solids since 1 month. She also gives history of choking sensation intermittently. She always sleeps in semirecumbent position. She was seen by surgeons and fine-needle aspiration cytology (FNAC) of lesion was performed, which showed poorly differentiated thyroid carcinoma.

Q. How do you define obesity?

Ans. Obesity is defined as a body weight that exceeds the expected or ideal weight by more than 10%, taking into account height, age, body built, and sex. An alternative definition is based on fat content of the body—patient is said to be obese when more than 25% of the body weight in males or 30% in females is attributable to fat (normally 15–18% body weight is fat). Morbid obesity is defined as actual weight exceeding twice the ideal body weight (IBW). **Table 1** gives division of patients according to body mass index (BMI) or Quetelet index. It was described by Lambert Adolphe Jacques Quetelet in 1835. It is calculated by dividing the patient's weight in kilograms by his height in meters square (**Table 1**).

$$\text{BMI} = \text{Weight (kg)} / [\text{Height (m)}]^2$$

TABLE 1: Categories of obesity.

BMI (kg/m ²)	Category
<18.5	Underweight
18.5–24.99	Normal
25–26.9	Overweight
27–30	Mild obesity
>30	Moderate obesity
>35	Severe obesity
>40	Morbid obesity
>50	Super obesity

(BMI: body mass index).

Q. How will you proceed with the preanesthesia check-up in this patient? What are the associated diseases linked to obesity, which can alter anesthesia management?

Ans. This patient's BMI is 42.6 kg/m² and she falls in the category of morbid obesity. Patients with morbid obesity have long list of comorbidities, which are of concern to the anesthesiologist (**Table 2**). Careful history should be taken to ascertain or rule out presence of comorbidities. When necessary, leading questions can be asked. The medication history also can be a guide to know patient's comorbidities. Always take detailed history to find out or

TABLE 2: Diseases linked to obesity.

System	Disease
Cardiac	Hypertension
	Atherosclerosis
Respiratory	Sleep apnea
	Asthma
	Pulmonary embolism
	Pulmonary hypertension
Endocrine	Obesity hypoventilation syndrome
	Diabetes, gout, and glomerulosclerosis
Hepatic	Fatty liver
	Cholelithiasis
Vascular	Varicose veins
	DVT
Skeletal	Degenerative arthritis and carpal tunnel syndrome
Oncology	Carcinoma endometrium
	Carcinoma breast
	Carcinoma prostate
	Carcinoma colon

(DVT: deep vein thrombosis).

rule out conditions that may not have been diagnosed, before morbidly obese patients are subjected to surgery and anesthesia. Most important among these are:

- *History of symptoms of diabetes mellitus (DM):* Ask for polyuria, polydipsia, nocturia, frequent fungal skin infections or recurring conjunctival, gum, or urinary tract infections, irritability and mood changes, blurred visions, tingling and numbness in legs, slow healing of cuts and wounds, etc.
- *History of airway involvement:* Habitual snoring, frequent interruptions in breathing during sleep as reported by others, excessive daytime sleepiness, hypertension, nocturnal choking, waking unrefreshed, and morning headaches.
- *Sleeping habits:* Many patients are unable to lie flat for several years, and may routinely sleep sitting up in an armchair. An assessment of the ability to tolerate the supine position may reveal profound oxygen desaturation, airway obstruction, or respiratory insufficiency.
- *Dietary habits:* It is also important to recognize patients with nutritional deficiency in the face of obesity, as most of the times these patients are on some sort of weight reduction diet, which may give rise to various nutritional deficiencies.
- *The drug history:* Ask if patient is consuming amphetamine-based appetite suppressants, as these contribute to increased perioperative cardiac risk.
- *Exercise tolerance:* Since morbidly obese patients have restricted physical activity, one cannot rely on exercise tolerance as measure of cardiorespiratory reserve. For cardiac assessment, follow American Heart Association (AHA) guidelines on perioperative cardiac evaluation for noncardiac surgery.

Q. How is cardiovascular system function altered with increasing weight?

Ans. Ischemic heart disease, hypertension, and cardiac failure are the most common occurrences with obesity. The adipose tissue has a resting blood flow of 2–3 mL/100 g/min, but it can increase up to 10-fold after meals. However, with increasing obesity, the perfusion per unit mass decreases. It falls from 2.36 to 1.53 mL/min when the percentage of fat increases from 20 to 36% of the body weight, and so the increase in cardiac output is not directly proportional to the total fat. Thus, each kilogram of fat acquired above IBW requires an increase in cardiac output of 20–30 mL. This increased cardiac output is mainly achieved through increase in stroke volume and not through increase in heart rate. The left ventricle dilates in response to this volume overload, and over time develops an eccentric type of hypertrophy to keep the wall stress normal. The

left atrium also enlarges in obese individuals and this is initially caused by the increased blood volume and venous return. Later, other factors like left ventricular hypertrophy (LVH) and diastolic dysfunction may also be responsible for increased left atrial size. Right ventricular structure and function may be similarly affected by the above-mentioned morphologic and hemodynamic alterations and by pulmonary hypertension related to the sleep apnea or obesity hypoventilation syndrome (OHS).

Arrhythmia: There is an increase in the incidence of sudden cardiac death and arrhythmias in obese. This may be due to increased levels of catecholamine. In addition, increased free fatty acid levels in the obese may also affect repolarization.

Coronary artery disease: Obesity is an independent predictor of coronary artery disease, as observed in many studies, and is due to accelerated atherosclerosis.

Hypertension: Blood pressure is the product of cardiac output and systemic vascular resistance, and cardiac output is increased in obese patients because of increased blood flow to the adipose tissue. Blood pressure increases by 6.5 mm Hg for every 10% increase in body weight. Generally, systemic vascular resistance should be low in obese individuals because of the increased cross-sectional area of the vascular bed. However, in addition to store fat, adipose tissue also acts as an endocrine organ and synthesizes variety of peptides and nonpeptide compounds like interleukin-6 (IL-6), plasminogen activator inhibitor-1, resistin, lipoprotein lipase, etc., which lead to low-grade inflammation and overactivity of the sympathetic nervous system. In addition, a disordered sleep pattern may also increase the systemic vascular resistance. Thus, with increasing severity of obesity, hypertension becomes more prevalent.

Q. What is obesity cardiomyopathy?

Ans. Cardiomyopathy in obese individuals is caused by a direct effect of obesity on the heart. Initially, various tissues of heart, like the sinus node, atrioventricular node, right bundle branch, and the myocardium, are infiltrated by fat cells. These can cause conduction defects like sinoatrial block, bundle branch block, and, rarely, atrioventricular block. Subsequently, such infiltration may cause pressure-induced myocyte degeneration. Accumulation of triglycerides in myocytes can directly cause cell dysfunction because of lipotoxicity. Adipose cells also secrete locally active molecules like adipokines, which indirectly damage adjacent myocytes. In addition, increased cardiac output and blood volume lead to ventricular dilatation and subsequently hypertrophy of left ventricle, which then can lead to diastolic dysfunction

(failure to relax properly). If the wall thickening fails to keep pace with chamber dilatation, systolic dysfunction (failure to contract properly) can occur as well. This disorder is called as obesity cardiomyopathy and can occur in absence of systemic hypertension and underlying organic heart disease. This is the reason why preoperative echocardiography is indicated in morbidly obese. The predominant causes of death in those with obesity cardiomyopathy are progressive congestive heart failure (CHF) and sudden cardiac death.

Q. Describe types of pulmonary involvements in morbid obesity. What is the difference between obesity hypoventilation syndrome (OHS) and obstructive sleep apnea (OSA)?

Ans. Along with cardiac disorders, various pulmonary disorders are of major concern to anesthetists. Most among these are OHS, OSA, and cor pulmonale. In addition, patients with morbid obesity usually have decreased pulmonary reserve even if they do not have specific pulmonary disorder. These patients also have an increased incidence of restrictive pulmonary disorder. Even if consequences of pulmonary dysfunction are less severe than cardiac, they are more common. Morbidly obese patients have reduced forced vital capacity (FVC), functional residual capacity (FRC), and total lung capacity (TLC) with decreased expiratory reserve volume and increased respiratory resistance. Due to these reasons, some amount of pulmonary insufficiency is always present in morbidly obese. These changes are of particular interest to the anesthetist, as added decrease in these values due to anesthesia, supine position, and postoperative pain, can worsen respiratory function in the postoperative period. Also, these patients have decreased chest wall compliance due to fat deposition on chest wall. All these changes lead to decreased respiratory reserve, difficulty in ventilation, and faster desaturation.

Obstructive sleep apnea: The presence of OSA indicates high chance of having other sequelae like hypertension, LVH, and right ventricular hypertrophy (RVH). Diagnosis is made based on polysomnography (PSG), commonly known as sleep study. OSA can be suspected from clinical symptoms and signs. History of symptoms, such as habitual snoring, interrupted nocturnal breathing during sleep as reported by others, excessive daytime sleepiness, arterial hypertension, nocturnal choking, waking unrefreshed, morning headaches, is frequently seen with OSA. Abnormal anatomy of the pharynx, increased collapsibility of the upper airway, and defective airway reflexes all lead to development of OSA. Obesity is usually associated with OSA due to the deposition of fat in the lateral pharyngeal

wall, resulting in decreased patency of the pharynx and increase in the risk of airway collapse, particularly during sleep, when there is relaxation of the pharyngeal dilator muscles. Decreased upper airway activity with rapid eye movement (REM) sleep also leads to pharyngeal narrowing. Vibration of upper airway structures due to turbulent flow results in snoring. Also, the pharynx tends to collapse due to Bernoulli effect, with partial or complete obstruction. Hypercapnia and hypoxia, which follow, lead to awakening and opening of pharynx due to increased upper airway muscle activity. The improved breathing decreases carbon dioxide tension and improves oxygen saturation. The cycle repeats with sleep onset. These patients are very sensitive to narcotics and sedatives. While many anesthetists are worried about induction and intubation in these patients, truly challenging period is extubation and postoperative period. It may be very difficult to balance postoperative pain relief and apnea. Patients given more narcotics during intraoperative period have high incidence of postoperative complications. These patients commonly have low baseline PaO_2 but oxygen supplementation does not improve PaO_2 , as they have longer periods of apnea and respiratory center is stimulated by hypoxia and not hypercarbia.

Obesity hypoventilation syndrome: OHS, also known as Pickwickian syndrome, is distinct from OSA and was originally described as combination of morbid obesity, hypersomnolence, plethora, and edema. While in OSA, patient breathes normally while awake, in OHS, patient is hypercapnic at rest as well as while awake. The cause of OHS is unknown but likely involves combination of disorder of respiratory center and decreased chest wall compliance. It is usually diagnosed when patient's BMI is more than 30 kg/m^2 , and they have resting hypercapnia (PCO_2 more than 45 mm Hg) in absence of other causes of hypoventilation, such as airway obstruction, drug overdose, neuromuscular disorders, etc.

Q. How does one diagnose OSA? What is polysomnography (sleep study)?

Ans. The STOP-BANG screening questionnaire in which one point is scored for each positive feature and a score ≥ 3 is high risk and ≥ 5 is a significant risk of OSA. It involves features like Snoring, Tiredness, Observation of stopped breathing or choking or gasping during sleep, high blood Pressure, $\text{BMI} > 35 \text{ kg/m}^2$, Age > 50 years, Neck circumference $> 43 \text{ cm}$ (17 in) for males, $> 41 \text{ cm}$ (16 in) for females and male Gender.

A score of ≥ 3 has shown a high sensitivity (90%) for detecting OSA but low specificity (28%) and a high positive predictive value of 85%. It is a validated tool to screen patients for OSA, spot unrecognized OSA, and can be used in identifying and risk-stratifying surgical patients.

Polysomnography is indicated for the diagnosis of sleep-related disorders. Standard PSG is performed in a sleep laboratory, hospital, or other dedicated unit under the supervision of a sleep technician. It includes measurements of O_2 saturation, electrocardiography (ECG), electroencephalography (EEG), electromyography (EMG), electrooculography (EOG), airflow, and respiratory effort measurements. Nasal and oral airflow is measured using pressure transducers or a thermocouple, fitted in or near the nostrils. The study identifies sleep architecture, number and degree of arousals, number and type of apneic episodes, episodes of oxygen desaturation and severity, cardiac arrhythmias, limb movements, disorders associated with REM sleep, and seizure activity.

Indications for sleep studies:

- Witnessed apnea during sleep greater than 10 seconds in duration, or
- Any combination of two or more of the following:
 - Excessive daytime sleepiness as evidenced by one or more of the following:
 - Inappropriate daytime napping (e.g., during driving, conversation, or eating)
 - Sleepiness that interferes with daily activities (the following should be ruled out as a cause for these symptoms—poor sleep hygiene, medication, drugs, alcohol, hypothyroidism, other medical diagnoses, psychiatric or psychological disorders, social or work schedule changes)
 - An Epworth Sleepiness Scale score (normal 0–9 and maximum 24) greater than 10
 - Persistent or frequent socially disruptive snoring
 - Obesity (BMI greater than 30 kg/m^2) or hypertension
 - Choking or gasping episodes associated with awakenings
 - Symptoms suggesting narcolepsy, e.g., sleep paralysis, hypnagogic hallucinations, and cataplexy
 - Violent or injurious behavior during sleep
 - Other situations (if nocturnal pulse oximetry suggests nocturnal oxygen desaturation)
 - Unexplained right heart failure
 - Unexplained polycythemia
 - Presence of or increase in cardiac arrhythmias during sleep
 - Unexplained pulmonary hypertension
- Excessive daytime sleepiness together with witnessed periodic limb movements of sleep
- Unusual or atypical parasomnias based on patient's age, frequency, or duration of behavior
- Patient's with moderate or severe CHF, stroke/transient ischemic attack (TIA), coronary artery disease, or

significant tachycardic or bradycardic arrhythmias who have nocturnal symptoms suggestive of a sleep-related breathing disorder or otherwise suspected of having sleep apnea.

Q. What is apnea-hypopnea index (AHI) and how do you grade it?

Ans. This is an index of total number of complete cessations (apnea) and partial obstructions (hypopnea) of breathing occurring per hour of sleep. These pauses in breathing last at least for 10 seconds and are associated with a decrease in oxygenation of the blood. In general, the AHI can be used to classify the severity of disease (mild 5–15, moderate 15–30, and severe greater than 30). It is also known as respiratory disturbance index or RDI. The effects of sleep apnea are not only functional and psychological but they also modify physiology. People with sleep apnea have increased sympathetic activity and endothelial and metabolic dysfunction. All this leads to higher incidence of cardiovascular complications. These patients are at increased risk for high blood pressure, ischemic heart disease, stroke, and CHF. As the AHI score increases, the risk of cardiovascular events increases. So, the AHI is a good indicator of disease severity and the urgency to treat a patient increases with increasing AHI.

Patient gives history of hypertension for last 9 years for which she is on amlodipine 5 mg twice daily and atenolol 25 mg once daily. On direct questioning, she says that she is regular with her treatment and follow-up and that her blood pressure remains on higher side of normal. She is also a diabetic being treated with oral hypoglycemic agents. She gives history suggestive of obstructive sleep apnea: history of snoring, history of witnessed apneic spells by husband at night, history of daily afternoon nap of 4 hours.

She fell 2 years ago and suffered from Potts fractures of left leg and tibial and patellar fracture of right knee. She had undergone surgery under GA and postoperative course was uneventful. Following fracture, she complains of stiff and painful right knee due to which she has restricted mobility, unsteady gait, and she walks with walker. Following this surgery, she had developed swelling in both legs, with darkening of skin and tingling in her both lower legs. She was investigated for this—compression venous ultrasound was performed, which was negative for deep venous thrombosis (DVT). She was started on pentoxifylline orally.

Q. How should a patient with morbid obesity be investigated for any surgery? What investigations would you advice for this patient?

Ans. Investigations should be tailored to the individual patient, depending on comorbidities and the type and urgency of surgery. A full blood count, electrolytes, renal and liver function tests, and blood glucose form a basic set

of investigations. Arterial blood gas (ABG) analysis will be useful in those suspected of respiratory comorbidity (OSA, OHS, large collar size, and other pulmonary disease). In many times, cause of obesity is hypothyroidism and from clinical history and examination, it is very difficult to rule out hypothyroidism. Hence, thyroid functions form a part of baseline investigations. As cardiac dysfunction of varying degrees is quite common in long-standing morbid obesity, a preoperative ECG is essential to look for significant rhythm disturbances, LVH and cor pulmonale, and as a guide to indicate need for more extensive cardiac investigation. Studies have shown that in patients with evidence of RVH or cor pulmonale, a period of elective nocturnal noninvasive ventilation before elective surgery is beneficial. Nocturnal noninvasive ventilation is effective in relieving right heart failure, daytime somnolence, and pulmonary hypertension. Chest X-ray may be used to assess cardiothoracic ratio and evidence of cardiac failure. Younger patients, those at the lower end of the BMI range, those with a good exercise tolerance, and those with a benign fat distribution, need not be tested further. A short walk along the ward or an attempt at climbing a flight of stairs can give useful functional information about effort tolerance. The first decision would be whether or not patient can exercise sufficiently to get 85% of predicted maximal heart rate. Some group of patients will require detailed preoperative cardiac evaluation. These patients include—patients with limited performance status, patients with hypertension, those with sleep apnea and hypoxia and hypertension, and those with history of edema feet/face. Patients with these features should undergo transthoracic echocardiography to estimate systolic and diastolic function, valvular status, and chamber dimensions, although good images may be difficult to obtain by the transthoracic technique. In patients in whom echocardiographic window is unsatisfactory, radionuclide ventriculography may be used to assess LV and RV morphology and function. A pharmacological stress test like dobutamine stress echocardiography or adenosine dipyridamole scan may be indicated, if more than or equal to three cardiac risk factors (history of heart disease, CHF, history of cerebrovascular disease, preoperative treatment of insulin, and preoperative creatinine >2 mg/dL) are present. Preoperative cardiopulmonary exercise testing can also be done to predict patients at increased risk of morbidity and mortality in some cases. If the noninvasive test is positive then invasive testing in the form of a cardiac catheterization is indicated.

Noting breath-holding time would help in assessing combined cardiorespiratory reserve.

Pulmonary function tests (PFTs) may reveal a restrictive defect, but are not performed on all patients unless there is a specific indication. Morbidly obese patients have reduced

FVC, FRC, and TLC with decreased expiratory reserve volume and increased respiratory resistance, and hence, one should expect some amount of pulmonary insufficiency in all morbidly obese. Studies have failed to show any impact of PFTs on perioperative outcome in obese patients, but can be used to predict risk of postoperative pulmonary complications.

Obesity is an independent risk factor for DVT and pulmonary embolism, and all the patients should be screened for presence of asymptomatic DVT using compression Doppler ultrasound.

Role of sleep studies in obese patients: In patients giving classical history of OSA, doing sleep studies may not add much to the preexisting knowledge. But in other obese patients, PSG might help in identifying population at risk. From present evidence, it is unclear whether routine PSG would improve safety and outcomes.

Since this patient has history of long-standing hypertension, diabetes, and OSA, she has high-risk for underlying cardiac dysfunction and would need cardiac workup in addition to ECG in the form of a transthoracic echocardiogram. Drug history (pentoxifylline) suggests that she might be suffering from peripheral vascular disease, which makes her a strong candidate for underlying coronary artery disease as well, which needs to be kept in mind. A dobutamine stress echocardiography should be asked for in this case.

This lady also has big neck mass and associated compressive symptoms, hence PFTs with flow-volume loop would help in identifying tracheal compression. Also, ABG should be done to know status of oxygenation. This patient also gives history of compressive symptoms (dysphagia); further investigation with CT neck and chest would help to know tracheal compression, deviation, and retrosternal extension. She also has very limited mobility; hence, chances of DVT are high. She should undergo lower limb compressive ultrasonography (USG) to detect presence of DVT as part of preoperative evaluation.

Potential venous and arterial access sites should be evaluated during the preoperative examination, and the possibility of invasive monitoring should be mentioned to the patient. Since this patient has such a strong history of OSA, patient should be told about possible need for postoperative elective mechanical ventilation.

Q. What are common ECG abnormalities associated with morbid obesity?

Ans.

- Low voltage complexes
- Left ventricular hypertrophy with/without strain
- Prolonged QT/QTc
- Inferolateral T-wave abnormalities

- Right axis deviation or right bundle branch block (RBBB)
- P-pulmonale.

The USG neck revealed multinodular goiter with left internal jugular vein (IJV) entrapment. CT scan showed enlarged thyroid, with tracheal and laryngeal compression, mediastinal extension of thyroid, compression of left common carotid artery, occluded left IJV, and presence of metastatic nodules in both lung fields. Hopkins mirror examination showed left vocal cord to be fixed. Her thyroid function tests, hematology, and biochemistry investigations were normal. ABG on air showed PaO_2 —69 mm Hg, SpO_2 —95%, and PaCO_2 —32 mm Hg. 2D-Echocardiography showed left ventricular ejection fraction (LVEF) of 60%, no regional wall motion abnormalities (RWMA), and severe PAH (PASP—65 mm Hg). Spirometry showed moderate restriction. Flow-volume loop did not reveal any significant tracheal compression. Barium swallow showed decreased distensibility of left pyriform fossa with filling defect.

Q. What is the role of preoperative weight reduction? Can this patient be put on weight-reducing diet before subjecting to surgery? What are the advantages and side effects of preoperative weight loss?

Ans. Even though weight reduction immediately before surgery has not been shown to reduce perioperative morbidity and mortality, it may reverse some of pathophysiological changes in the obese patients. Even mild weight reduction before surgery may reduce liver size facilitating surgical approach through diaphragmatic hiatus (for bariatric surgery). In addition, decrease in intra-abdominal pressure could improve conditions for mechanical ventilation.

For preoperative weight reduction, it is important that adequate proteins should be provided (1.5 g/kg IBW), so that lean body mass will be maintained. Diets in preoperative period should be properly supervised, as it can dangerously influence perioperative outcome. Starvation diets, low- and very low-calorie diets, and protein-deficient diets can lead to development of severe cardiac arrhythmias and even sudden death. The implementation of low fat and low-calorie diet typically results in an increased intake of vegetables and low intake of milk and dairy products, cereals, fruits, meat products, and added fats. This may result in a significant decreased daily intake of phosphorous, zinc, magnesium, iron, as well as vitamins B_1 , B_2 , and B_6 . All these deficiencies, particularly B complex, should be replenished before surgery. However, reduction in simple carbohydrates reduces glycemia and lipogenesis, and should be routinely recommended. A particularly compromised group is patients who have had prior bariatric surgery. They must always be considered to have latent malnutrition and thus they present a greater risk for postoperative complications.

Since this patient has a large thyroid with compressive symptoms and malignancy, she is not a candidate for preoperative weight reduction and surgery should be conducted as soon as possible. However, a dietician should be involved in her perioperative management and a diet modification can be done.

Q. How can we stratify the risk of surgery in the morbid obese patients?

Ans. The Obesity Surgery Mortality Risk Stratification score (OS-MRS) has been validated for patients undergoing gastric bypass surgery to identify the risk factors associated with mortality. It includes features of metabolic syndrome and sleep-disordered breathing. Although only validated for bariatric surgical patients, it may be applicable to obese patients undergoing nonbariatric operations.

The Obesity Surgery Mortality Risk Stratification score:

- BMI $>50 \text{ kg/m}^2$ —1
- Male—1
- Age >45 years—1
- Hypertension—1
- Risk factor for pulmonary embolism—1:
 - Previous venous thromboembolism (VTE)
 - Vena cava filter
 - Hypoventilation (sleep-disordered breathing)
 - Pulmonary hypertension
 - Poor mobility

Risk of mortality:

- Class A: 0–1 points 0.2–0.3%
- Class B: 2–3 points 1.1–1.5%
- Class C: 4–5 points 2.4–3.0%.

Q. How will you prepare this patient for surgery? What premedication would you use?

Ans.

- An important adjunct to dietary management is implementation of physiotherapy and exercise program. This improves glucose tolerance and respiratory capacity. Breathing exercises and incentive spirometry should be a routine practice in order to improve perioperative oxygenation and prevent postoperative atelectasis.
- No sedatives or narcotics should be used. Use counseling and reassurance technique. Patients with high level of anxiety can be given very light premedication but avoid narcotics and monitor them carefully.
- This patient has very strong history of obstructive sleep apnea. Also, she has a large thyroid with mediastinal extension. Both these factors make her very prone to mechanical airway obstruction in the perioperative period. These patients are extremely sensitive to sedatives and analgesics, and may develop apnea or respiratory obstruction following even small doses of

these drugs. Hence, premedication should be avoided in this patient. She should be observed on previous night for frequency of obstruction and periods of desaturation.

- Obesity is a risk factor for DVT. The adjusted risk ratio of PE according to weight increases from 1.0 in patients with BMI less than 25 kg/m² to 2.7 in patients with BMI more than 40 kg/m². DVT prophylaxis should be initiated before the induction of anesthesia. Unfractionated heparin, 5,000 units subcutaneously, administered before surgery and then every 8–12 hours until the patient is fully mobile, has been shown to reduce the risk of DVT. Also, low-molecular weight heparins, such as enoxaparin, have been used for thromboembolism prophylaxis. Additionally, appropriately sized compression stockings and use of pneumatic sequential compression devices intraoperatively and postoperatively help in reducing risk of DVT.
- Antacid prophylaxis, as these patients are prone for aspiration. A significant residual gastric volume and acidic pH is common. Antacids, proton-pump inhibitors, H₂ receptor antagonists, and prokinetic agents are all likely to be of value in the perioperative period. Routine prophylaxis with ranitidine or a proton-pump inhibitor is advisable and can be administered orally at the time of premedication. In obese patients, not at risk of aspiration, standard American Society of Anesthesiologists (ASA) guidelines of fasting are followed. Lately, gastric ultrasound has been utilized in clinical trials to assess gastric volume. In one small study in severely obese patients, gastric ultrasound estimated gastric volume correlated well with volume of gastric aspirate, with accuracy similar to that reported for gastric ultrasound in nonobese patients. But, results have not been correlated with the risk of aspiration during anesthesia.
- Preoperative antibiotics prophylaxis as recommended for all surgical procedures.
- Avoid IM injections due to unpredictable absorption.

Q. What are the principles of anesthetic management? What precautions would you take while padding and positioning?

Ans. Operation theater (OT) tables have weight limits and overweight patients can lead to table breakage. Whenever possible, use table with proper weight-bearing capacity. If that is not available, two OT tables (side by side, with a board across the lower half so the back can still flex to a sitting position) should be used. Patients are frequently wider than table and positioning an arm by the side may be difficult. Due to the layer of fat around arm and back, patient's shoulders are frequently far above the table and

arm boards, making positioning of arms on arm boards uncomfortable for patient, and fastening the arms under tension under anesthesia will put brachial plexus at risk of hyperabduction injury. To prevent this, extra padding may be required on arm board.

Operation table should be powered, as manual operating tables become difficult to manage. Standard straps may not reach around patients and may make patient prone to fall.

Obese patients are also more prone for pressure-related injuries and proper padding of buttocks and back is necessary to prevent gluteal compartment syndrome, which can lead to rhabdomyolysis and renal failure. Patients with surgery longer than 4 hours are prone to develop this syndrome. Prone position is poorly tolerated by these patients and lateral decubitus is better because it keeps abdominal weight off chest. Postoperative bed should have higher weight limit than that used for normal patients.

This patient was scheduled to undergo total thyroidectomy. Clinical examination revealed morbidly obese patient with orthopnea (she could not lie supine in bed). She was receiving oxygen via nasal cannula at 3 L/min. Airway assessment showed short bulky neck with Mallampati class IV. Thyromental distance could not be estimated due to the tumor size and due to limited neck movements. The trachea could not be palpated anteriorly except for 1 inch at the cricoid level.

Q. Why and how should one preoxygenate these patients?

Ans. There are two techniques advocated for preoxygenation—breathing 100% oxygen through well-fitting facemask for 3 minutes or taking 4 vital capacity breaths with 100% oxygen. The purpose of preoxygenation is to fill lungs with 100% oxygen and thus have prolonged apnea time safely. Studies have shown that breathing 100% oxygen for 3 minutes is superior to 4 vital capacity breaths. Rate of desaturation will depend on FRC and BMR. Thus, obese patients would desaturate faster, as they have very reduced FRC. Rate of desaturation is fastest for morbidly obese, followed by children and septic patients due to increased BMR, followed by normal adults. As incidence of difficult intubation is high with morbid obesity, one can use continuous oxygen insufflation using nasopharyngeal catheter at the rate of 15 L/min during laryngoscopy (apneic oxygenation). Apnea time of patients with difficult airways, including several obese patients undergoing general anesthesia for upper airway operations, was extended with continuous delivery of O₂ by “transnasal humidified rapid-insufflation ventilatory exchange” (THRIVE), which combined the benefits of apneic oxygenation with continuous positive airway pressure (CPAP) and gas exchange through flow-dependent dead-space flushing. It is important to remember for successful apneic oxygenation,

head elevation, jaw thrust, nasal prongs, or an oral airway may be required. Some recently marketed blades have provision for continuous administration of oxygen during laryngoscopy.

Q. Why do you prefer to induce morbidly obese patients in reclining position? Why do these patients tolerate supine position badly?

Ans. Functional residual capacity is the sum of ERV (the maximum volume expired after passive expiration) and RV (the volume left in the lungs after maximal expiratory efforts). The closing volume (CV) is the lung volume above RV at which airway collapse occurs during expiration, mainly in the lower lung zones due to gravitational effects. The relation between CV and FRC is determined by the balance between the inward elastic recoil of lungs and outward pull by thoracic cage. In severe obesity, CV exceeds ERV due to a decrease in ERV. Some airways are closed during tidal breathing resulting in increase in shunt and arterial hypoxemia. The airway closure becomes more pronounced when these patients assume supine position since the increased weights of chest wall and abdomen exacerbate early airway closure and promote arterial hypoxemia.

Q. How would you induce this patient and how will you manage her airway?

Ans. A theater table with an appropriate maximum weight allowance must be used. There must be enough trained and experienced staff in theater to assist with moving the patient quickly, should it become necessary during induction. Standard monitoring should include a correct-sized blood pressure cuff. Venous cannulation can sometimes be difficult and central venous cannulation may be necessary. Direct intra-arterial monitoring should be considered for situations where rapid hemodynamic changes are possible, surgery is prolonged, and in patients with cardiorespiratory disease or if noninvasive arterial pressure monitoring is impractical.

Airway management: The choice between awake and asleep intubation is difficult and depends on anticipated difficulties along with experience of anesthetist. Obesity is associated with increased incidence of difficult mask ventilation and difficult intubation. Commonly used anesthetic agents like propofol, thiopentone, narcotics, benzodiazepines, and muscle relaxants can all lead to pharyngeal collapse. Obese patients therefore should be carefully evaluated for difficult airway. History of sleep apnea, limited neck movements, limited mouth opening, large tongue, and short thyromental distance all increase the risk for difficult airway.

Also in these patients, gastric insufflation during ineffective mask ventilation will further increase risk of regurgitation and aspiration of stomach contents. Hence,

fiber-optic intubation is frequently recommended in these patients. Also, alternative plan of securing the airway should be ready in case first plan fails. An emergency tracheostomy is challenging in these patients and may not be practical or safe, which further favors awake intubation. Looking at history and clinical examination, this patient should undergo awake fiber-optic bronchoscopy (FOB). Patient positioning is of paramount importance before induction, particularly head position. A "sniffing the morning air" position may be difficult to achieve due to the large soft tissue mass of the neck and chest wall, and a wedge or blanket beneath the shoulders is often beneficial ("ramped" technique). Difficulties encountered in bag and mask ventilation can be overcome by a four-handed technique. Using anesthesia ventilator and positive end-expiratory pressure (PEEP) for mask ventilation helps in maintaining FRC and thus improves oxygenation.

Standard use of the 30° reverse Trendelenburg (head up) position during preoxygenation, induction, and emergence from anesthesia is useful. A polio handle and/or a long blade may be helpful in intubation in case direct laryngoscopy is planned. Difficult airway management cart should be ready. Efficient help in form of an additional trained anesthetist, operating room technician, and the surgeon is mandatory during induction and emergence. Ultrasound may be used to assist in vascular access, nerve block, and neuraxial procedures.

Q. What would be your choice of inhalational agent?

Ans. For faster induction and rapid recovery, volatile agents should be insoluble in blood with minimal or no metabolic degradation and low lipid solubility. Rapid elimination and analgesic qualities of nitrous oxide make it a preferred agent. Isoflurane is cheaper compared to desflurane and sevoflurane, but it has a longer washout period and produces slower awakening as compared to other two agents. Desflurane has low partition coefficient (0.42) that allows for faster washout and elimination than sevoflurane and it has even been observed with BIS monitoring that desflurane produces faster awakening. However, sevoflurane may be the preferred agent, as it causes less tachycardia, which is advantageous in patients with underlying CAD. So, one has to decide the agent of choice depending on whether your patient has strong history of OSA. If you want faster awakening, choose desflurane. If your patient has bad CAD, you want better rate control then choose sevoflurane.

Q. How would you manage this patient intraoperatively? What are the ASA task force guidelines for patients with OSA?

Ans. Because of their propensity for airway collapse and sleep deprivation, patients with OSA are especially susceptible to

the respiratory depressant and airway effects of sedatives, opioids, and inhaled anesthetics. Therefore, in selecting intraoperative medications, the potential for postoperative respiratory compromise should be considered. Select short-acting agents (e.g., alfentanil, propofol, midazolam, and atracurium), and avoid using long-acting agents (e.g., morphine, diazepam, or pancuronium).

For superficial procedures, one should consider the use of local anesthesia or peripheral nerve blocks, with or without moderate sedation. Use of ultrasound and nerve locator adds to safety and success of the procedure. If moderate sedation is used, ventilation should be continuously monitored by capnography, if feasible, because of the increased risk of undetected airway obstruction in these patients. Consider administering CPAP during sedation to patients. General anesthesia with a secure airway is preferable to deep sedation without a secure airway, particularly for procedures that may mechanically compromise the airway. For all but very short surface procedures, intermittent positive pressure ventilation (IPPV) will be needed. High inflation pressures will be required due to decreased compliance and increased resistance. ETCO_2 monitor would guide regarding adequacy of ventilation. Dose of muscle relaxants should be guided by monitoring with peripheral nerve stimulator. Unless there is a medical or surgical contraindication, patient at increased perioperative risk from OSA should be extubated while awake. Full reversal of neuromuscular block should be verified before extubation. When possible, extubation and recovery should be carried out in the lateral, semi-upright, or other nonsupine position.

Consider central neuraxial blockade (spinal/epidural) for peripheral procedures. Postoperative shivering increases oxygen consumption and cardiovascular stress. Effective temperature maintenance during perioperative period is important; it also reduces postoperative wound infection. Forced warm air blankets in combination with fluid warmers are effective in preventing perioperative hypothermia.

Q. What intraoperative monitoring would you institute?

Ans. Other than standard intraoperative monitoring of ECG, SpO_2 , temperature, and ETCO_2 , these patients require invasive monitoring frequently. Invasive hemodynamic monitoring is indicated in patients with CHF, renal insufficiency, or severe pulmonary hypertension (PA systolic >60–70 mm Hg) or for procedures with expected large fluid shifts perioperatively. A preoperative echocardiography will be of great help to decide on indications for invasive hemodynamic monitoring.

Appropriate sized noninvasive blood pressure cuff is important for accurate BP measurement. Usual sized BP

cuffs are too small and larger cuffs (thigh cuffs) are too wide and extend down past cubital fossa in most of the patients. Also, most of these patients have conical-shaped arms that are difficult to keep a BP cuff on. Many clinicians prefer routine invasive arterial pressure monitoring, as noninvasive pressure may be unreliable due to lack of adequate sized cuff. Intraoperative fluid balance is difficult to assess. The blood volume may be reduced from normal 70 mL/kg. Central venous pressure (CVP) monitoring is helpful with large blood losses, but again one has to consider underlying cardiac dysfunction, RVH, or cor pulmonale, where CVP will lose its significance as an indicator of preload. Urine output may be a satisfactory measure of adequate circulating volume when CVP cannot be monitored. For major surgeries, one may consider use of advanced hemodynamic monitoring techniques of measuring continuous cardiac output like PiCCO or FloTrac, which also gives reliable information of preload by giving stroke volume variation (SVV) or pulse pressure variation (PPV) during controlled mechanical ventilation.

Q. What are the principles of postoperative management according to the ASA task force guidelines for patients with OSA?

Ans. Where possible, those patients fit enough for extubation should be extubated wide awake in the sitting position and transferred to an appropriate postoperative environment. Regional analgesic techniques should be considered to reduce or eliminate the requirement for systemic opioids in patients with increased perioperative risk from OSA. If neuraxial analgesia is planned, weigh the benefits (improved analgesia and decreased need for systemic opioids) and risks (respiratory insufficiency from cranial spread) of using an opioid or opioid–local anesthetic mixture as compared with a local anesthetic alone. If patient-controlled systemic opioids are used, continuous background infusions should be avoided. Nonsteroidal anti-inflammatory drugs (NSAIDs) and other modalities like transcutaneous electrical nerve stimulation (TENS) should be considered, if appropriate to reduce opioid requirements. Avoid concurrent administration of sedative agents like benzodiazepines, as it increases the risk of respiratory depression and airway obstruction. In morbid obesity, acetaminophen (paracetamol) should be used in standard doses, as its volume of distribution is largely confined to the central compartment. However, as its clearance is increased in obesity, consider increasing frequency of dosing when analgesia is unsatisfactory.

Supplemental oxygen should be administered continuously to all patients who are at increased perioperative risk from OSA, until they are able to maintain their baseline oxygen saturation while breathing

room air. One has to keep in mind that supplemental oxygen may increase the duration of apneic episodes and may hinder detection of atelectasis, transient apnea, and hypoventilation by pulse oximetry. CPAP or nasal intermittent positive pressure ventilation (NIPPV), with or without supplemental oxygen, should be continuously administered when feasible (e.g., when patients are not ambulating) to patients who are already using these modalities preoperatively, unless contraindicated by the surgical procedure. Encourage patients to bring their own equipment to the hospital, as it will improve compliance.

If possible, patients at increased perioperative risk from OSA should be placed in nonsupine position (lateral or head elevation) throughout the recovery process. Hospitalized patients, who are at increased risk of respiratory compromise from OSA, should have continuous pulse oximetry monitoring even in ward. Continuous monitoring should be provided during entire period of hospitalization. Intermittent pulse oximetry or continuous bedside oximetry without continuous observation does not provide the same level of safety. If frequent or severe airway obstruction or hypoxemia occurs during postoperative monitoring, initiation of nasal CPAP or NIPPV should be considered. Early mobilization is encouraged where possible, as it reduces postoperative atelectasis and the risk of VTE.

Q. Describe role of perioperative NIV.

Ans. Patients with OSA generally are on CPAP or bi-level positive airway pressure (BiPAP). CPAP treats apneas and hypopneas by providing air under positive pressure through a nasal or facial mask, thus creating a pneumatic splint in the pharynx, which prevents collapse of the pharyngeal airway. A second theory postulates that noninvasive ventilation improves respiratory system compliance by reversing microatelectasis of the lung, thereby diminishing daytime work of breathing. BiPAP gives two levels of pressure—lower and higher alternatingly, and is sometimes better tolerated by the patients who do not tolerate CPAP. It is important to know what mode patient is on and what pressures being were being used, so that same settings can be used perioperatively. Also, since mask fit may be the problem, ask patient to get his own mask and head gear. Postoperative NIV allows patients to receive slightly higher doses of analgesics and gives a safety margin, as very frequently these patients desaturate to 70–80%, even with supplemental oxygen. This is especially true for patients with OSA and OHS, where it can lead to longer recovery stay and also hospital stay.

Q. What are intermittent pneumatic calf compression devices (IPCDs)? What are the indications and contraindications for their use?

Ans. An IPCD consists of a pair of double-walled, vinyl pneumatic sleeves is placed around the calves and connected to a compressor that inflates and deflates the garments. Compressions last 12 seconds per minute, with inflation pressure of 40 mm Hg. The leg sleeves (12–16 inches long) extend distally from the inferior border of the patella. They are applied preoperatively and are worn during surgery; they are removed once the patient begins walking. This device compresses the lower leg, reproducing the action of the calf muscles, thus promoting venous return. Studies indicate that calf compression stimulates fibrinolysis and contributes in preventing thrombus formation.

Intermittent pneumatic calf compression devices are indicated in patients prone for DVT; hypercoagulable states like malignancies, in prolonged surgeries leading to stasis of blood and in patients with contraindications for anticoagulation, requiring short-term DVT prophylaxis.

Contraindications to IPCD use include acute thrombophlebitis, suspected DVT, CHF, pulmonary edema, and leg ischemia due to severe peripheral vascular disease.

Q. How does bariatric surgery help morbidly obese individuals?

Ans. The precursors for obesity are multifactorial. They include genetic tendency, environmental effects, education, sex, race, and socioeconomic status. Obesity is mainly due to changes in behavior pattern, including decreased physical activity and overconsumption of high-fat food. There are simple solutions to the problem—education, sensible long-term diet, increased physical activity and exercise, and, in some cases, medication. Unfortunately though the answers are easy, they are not always practical and hence the only treatment that is effective is bariatric surgery. Following the weight loss, there is a high cure rate for diabetes and sleep apnea with significant improvement in other complications of obesity such as hypertension and osteoarthritis.

The two types of weight reduction surgeries available are—(1) gastric restrictive procedures like stapled gastropasty, Roux-en-Y gastric bypass, and adjustable gastric banding, and (2) malabsorptive procedures like biliopancreatic bypass and jejunoileal bypass. Some of these procedures have been discontinued either because of ineffective weight loss, as in horizontal gastropasty, or because of complications, as in jejunoileal bypass. Other techniques, such as gastric bypass and laparoscopic adjustable gastric banding (LAGB) have been widely adopted as the procedures of choice in weight reduction. Cardiopulmonary and wound complications remain a major morbidity problem in morbidly obese patients following open surgery. Obese patients can benefit greatly from minimally invasive techniques that may help to

reduce the incidence of these complications. Laparoscopic adjustable gastric banding, which was introduced in early 1990s, is now the most common procedure and offers the advantages of minimally invasive surgery, adjustability, and reversibility.

Q. What is the role of regional anesthesia in morbidly obese patient?

Ans. Although the superiority of epidural anesthesia in obese patients is not yet proven, a recent study has shown less postoperative reduction in vital capacity and other spirometry values and quick recovery of lung volumes in patients receiving epidural compared with those treated with opioids. Wherever it is possible and feasible, regional anesthesia should be administered for obese patients. The advantages are reduction in the use of opioids and inhalational agents, reduced postoperative complications, and less airway-related complications.

Main problem with the regional blocks is the technical challenge. Challenges include the lack of palpable bony landmarks, the depth of the space, and “false” loss-of-resistance in fatty tissues. Special long epidural and spinal needles may be needed. Use of ultrasound for identification of the epidural space and peripheral nerves improves safety and success of regional anesthesia. Insulated needles and a nerve stimulator can be used to identify the appropriate nerves for peripheral nerve blocks. Neuraxial spread of local anesthetics is directly related to BMI. Increased abdominal pressure shifts blood from the inferior vena cava into the epidural venous system, decreasing the volume of the epidural and subarachnoid spaces. Epidural fat further reduces the capacity of the epidural space resulting in higher blocks. For epidural and spinal blocks, local anesthetic dose requirements are reduced by 20–25%. Blocks extending above T5 can cause cardiorespiratory collapse and all resuscitation equipment should be kept ready.

Q. Enumerate the factors affecting drug doses (pharmacokinetics) in obese patients.

Ans. Classical pharmacokinetic parameters such as volume of distribution (Vd), clearance (Cl), and protein binding can change for some drugs in morbidly obese patients.

Volume of distribution is affected in obese patients due to decreased fraction of total body water, increased adipose tissue, increased lean body mass, altered tissue protein binding, increased blood volume and cardiac output, increased concentration of free fatty acids and cholesterol, and organomegaly.

Plasma protein binding is changed leading to the following—adsorption of lipophilic drugs to lipoproteins so increased free drug available, plasma albumin unchanged, and increased α_1 -acid glycoprotein.

Drug clearance is affected by—increased renal blood flow, increased GFR, increased tubular secretion, and decreased hepatic blood flow in congestive cardiac failure.

Highly lipophilic drugs such as barbiturates and benzodiazepines show significant increase in Vd for obese individuals. Less lipophilic compounds have little or no change in Vd with obesity. Exception to this rule is remifentanyl, which is a highly lipophilic but shows no significant change in distribution in obese individuals. Consequently, the absolute Vd remains relatively unchanged and the dosage should be calculated on the basis of IBW. Drugs with weak or moderate lipophilicity can be dosed on the basis of IBW or more accurately on lean body mass (LBM). These values are not identical because 20–40% of an obese patient's increase in total body weight (TBW) can be attributed to an increase in LBM. Adding 20% to the estimated IBW dose is sufficient to include the extra lean mass. Nondepolarizing neuromuscular blocking agents can be dosed in this manner.

Succinylcholine is an exception; dosage should be calculated using TBW. The majority of anesthetic drugs are strongly lipophilic. Increased Vd is expected for lipophilic substances, but this is not consistently demonstrated in pharmacological studies, because of factors such as end-organ clearance or protein binding. It has been observed that the Vd of water-soluble agents is less affected by obesity than lipophilic compounds (**Table 3**).

TABLE 3: Commonly used anesthetic agents and their doses.

Drug	Recommended dosing
Propofol	• Induction: IBW • Maintenance: TBW or IBW + (0.4 × excess weight)
Fentanyl	TBW
Thiopental	7.5 mg/kg IBW TBW
Midazolam	• TBW for initial dose • IBW for continuous dose
Vecuronium	• IBW
Atracurium	• TBW • Initial dose 0.15 mg/kg–2.3 mg/10 kg >70 kg • Supplemental dose 0.15 mg/kg–0.7 mg/10 kg >70 kg
Cisatracurium	TBW
Rocuronium	IBW
Succinylcholine	• TBW • >140 kg. Maximum 120–140 mg
Mivacurium	TBW: Divided dosing 0.15 mg/kg + after 30 seconds 0.15 mg/kg
Neostigmine	TBW
Alfentanil	• IBW or corrected weight • Corrected weight = IBW + (0.4 × excess weight)

Contd...

Contd...

Drug	Recommended dosing
Sufentanil	TBW, corrected weight BMI >40
Remifentanyl	IBW
Morphine	IBW
Paracetamol	IBW

(IBW: ideal body weight; TBW: total body weight).

Q. Enumerate treatment modalities for OSA.

Ans. Treatment modalities available are:

- **Mandibular advancement devices:**
 - Surgery—jaw advancement, uvulopalatopharyngoplasty, bariatric surgery, and tonsillectomy in children
 - Tracheostomy is curative but rarely used because of the associated morbidity.

Q. Enumerate the problems and their management in obese obstetric patients.

Ans. The problems are:

- Increased risk of chronic hypertension, pregnancy-induced hypertension (preeclampsia), and diabetes (two- to eightfold increase in incidence).
- A higher incidence of induced labor, assisted deliveries, and cesarean section.
- Weight gain and maternal diabetes may increase the incidence of fetal macrosomia (a newborn with an excessive birth weight), with associated risks and difficulty in delivery.
- Possibility of greater blood loss during cesarean section, the surgery tends to be longer, and the incidence of postoperative complications tends to be higher.
- Increased risk of anesthesia-related maternal morbidity and mortality during cesarean section, when compared with nonobese patients.
- Increased risk of fetal morbidity and mortality.
- Cephalad retraction of panniculus (layer of fat tissue, consisting of subcutaneous fat in the lower abdominal area) in morbidly obese during cesarean section may lead to hypotension and fetal compromise, as well as problems in gas exchange.
- In case of central neuraxial blockade, higher levels can lead to loss of intercostal muscle function, which may create greater breathing problems in the obese parturient.
- Supine and Trendelenburg positions given during cesarean section may further decrease FRC, increasing the likelihood of hypoxemia and aspiration.
- Use of PEEP to increase oxygenation may decrease cardiac output and possibly compromise uterine blood flow.

Problems with regional anesthesia are:

- Greater cephalad spread of local anesthetics during spinal anesthesia.
- The consequences of excessive blockade can create trouble with spinal in an obese patient with a difficult airway.
- Increased surgical time of cesarean section should be a consideration when selecting an anesthetic for spinal anesthesia.
- Higher incidence of failed epidurals in the morbidly obese due to technical problems.

Advantages of selecting an epidural are:

- **Slower onset:** Ability to titrate and less hypotension
- Potential for less motor blockade
- Facilitates postoperative analgesia.

Considerations for GA are:

- Higher risk of aspiration combined effect of pregnancy, obesity, and difficult airway.
- Anticipate a difficult bag and mask ventilation and laryngoscopy secondary to large breasts, poor neck range of motion, and a decreased chin to chest distance.
- **The mother's well-being should be considered first:** A rapid sequence induction should not be attempted, if the intubation is anticipated to be difficult. Opt for awake FOB-guided intubation.
- Consider use of a short-handled laryngoscope.
- Anticipate a more rapid oxygen desaturation than that in nonobese and nonpregnant obese patients.
- Preoxygenate well with 100% oxygen for 3 minutes of tidal ventilation.
- Anticipate difficulty in securing emergency airway via cricothyrotomy or tracheostomy, secondary to poorly defined landmarks and difficulty in proper positioning.
- Postoperative hypoxemia is more severe in obese patients, and the incidence is increased with a vertical incision.
- Patient should be recovered in semirecumbent position and provide supplemental oxygen administration.
- Women are at increased risk of VTE during pregnancy and the postpartum period. This risk increases further with obesity. Postoperative anticoagulation should be considered for these patients, but be careful about risk of postpartum hemorrhage.

Q. If a morbid obese patient has to undergo procedure under monitored anesthesia care or sedation, what are the anesthetic concerns?

Ans. The preoperative evaluation for patients undergoing sedation should be similar to those having general anesthesia. Airway should be evaluated properly and all airway equipment should be ready along with plan to secure airway, as patients with sleep-disordered breathing

are likely to have airway obstruction with even minimal sedation. Obese patients are not suitable for solo operator sedation procedures.

Q. What are the anesthetic concerns, if the patient has to undergo emergency surgery under general anesthesia?

Ans. Emergency surgery in obese patients should be managed by an experienced anesthetist in the care of the obese, along with an experienced surgeon in order to minimize the operative time and the risk of complications. Postoperatively, these patients will require proper monitoring and admission to ICU or high dependency unit, as they are at higher risk of emergency surgery complications. It may be difficult to examine abdomen and may have to rely on vital signs like tachycardia or new onset of abdominal pain or unexplained fever for diagnosing signs of intra-abdominal sepsis and may require ABGs and serum lactate for helping in diagnosis.

Q. What are the anesthetic concerns, if a morbid obese patient is posted for day care surgery?

Ans. The current guidelines from most anesthesia societies advocate automatic acceptance for day surgery of patients with BMI <40 kg/m² who do not have major comorbidity or comorbidities optimized prior to surgery or they being treated as day case will not alter perioperative risk. But for those with comorbidities or BMI >40 kg/m², decision to perform day care procedure should be based on proper patient evaluation and individualized discussion with patient and surgeon. The Society for Ambulatory Anesthesia suggests patients with a known diagnosis of OSA can be considered for day surgery, if they have and are able to use a CPAP device after discharge, if any comorbid conditions are optimized, and if postoperative pain relief can be provided predominantly by nonopioid analgesics.

Q. A morbid obese patient has a cardiac arrest on operating table. What are the problems anticipated in cardiopulmonary resuscitation (CPR)?

Ans. The major concerns in CPR of a morbid obese patient are:

- Difficulties in placement of defibrillator pads
- Establishment of vascular access
- Securing an effective airway causing a delay
- Quality of chest compressions delivered, efficacy of vasoactive drugs or the efficacy of defibrillator shocks may be affected
- Inspiratory airway pressures are higher than normal due to low chest wall compliance and may lead to excessive leak with supraglottic airway devices that may necessitate interruptions in chest compressions to allow effective ventilation even with advanced airway device

in place (i.e., a standard 30:2 compression-ventilation ratio).

Even though obese patients may have increased thoracic impedance, recommended defibrillation energies remain same as for other patients. If defibrillation remains unsuccessful, the defibrillator pads should be repositioned and the shock energy increased to the maximum setting. In case of difficult IV access, the intraosseous route for drug delivery is recommended. The upper humerus is a well-established point of access, and drug delivery during resuscitation is effective via this route. Standard doses of adrenaline and amiodarone should be used.

Summary of 2015 recommendations of Association of Anaesthetists of Great Britain and Ireland (AAGBI) and Society for Obesity and Bariatric Anaesthesia (SOBA).

1. Every hospital should nominate an anesthetic lead for obesity.
2. Operating lists should include the patients' weight and BMI.
3. Experienced anesthetic and surgical staff should manage obese patients.
4. Additional specialized equipment is necessary.
5. Central obesity and metabolic syndrome should be identified as risk factors.
6. Sleep-disordered breathing and its consequences should always be considered in the obese.
7. Anesthetizing the patient in the operating theater should be considered.
8. Regional anesthesia is recommended as desirable but is often technically difficult and may be impossible to achieve.
9. A robust airway strategy must be planned and discussed, as desaturation occurs quickly in the obese patient and airway management can be difficult.
10. Use of the ramped or sitting position is recommended as an aid to induction and recovery.
11. Drug dosing should generally be based upon lean body weight and titrated to effect, rather than dosed to TBW.
12. Caution is required with the use of long-acting opioids and sedatives.
13. Neuromuscular monitoring should always be used whenever neuromuscular blocking drugs are used.
14. Depth of anesthesia monitoring should be considered, especially when total intravenous anesthesia is used in conjunction with neuromuscular-blocking drugs.
15. Appropriate prophylaxis against VTE and early mobilization are recommended since the incidence of venous thromboembolism is increased in the obese.
16. Postoperative intensive care support should be considered, but is determined more by comorbidities and surgery than by obesity per se.

SUGGESTED READING

1. Association of Anaesthetists of Great Britain and Ireland Society for Obesity and Bariatric Anaesthesia. Peri-operative management of the obese surgical patient. *Anaesthesia*. 2015;70(7):859-76.
2. Carretero OA, Oparil S. Essential hypertension: Part I: Definition and etiology. *Circulation*. 2000;101(3):329-35.
3. Dixon JB, Schachter LM, O'Brien PE, Jones K, Grima M, Lambert G, et al. Surgical vs conventional therapy for weight loss treatment of obstructive sleep apnea: a randomized controlled trial. *JAMA*. 2012;308(11):1142-9.
4. Ingrande J, Lemmens HJ. Dose adjustment of anaesthetics in the morbidly obese. *Br J Anaesth*. 2010;105(1):i16-i23.
5. Mannarino MR, Di Filippo F, Pirro M. Obstructive sleep apnea syndrome. *Eur J Intern Med*. 2012;23(7):586-93.
6. Pelosi P, Gregoretti C. Perioperative management of obese patients. *Best Pract Res Clin Anaesthesiol*. 2010;24(2):211-25.
7. Poirier P, Giles TD, Bray GA, Hong Y, Stern JS, Xavier F. Obesity and cardiovascular disease: Pathophysiology, evaluation, and effect of weight loss. An Update of the 1997 American Heart Association Scientific Statement on Obesity and Heart Disease. From the Obesity Committee of the Council on Nutrition, Physical Activity, and Metabolism. *Circulation*. 2006;113(6):898-918.
8. Thompson J, Bordi S, Boytim M, Elisha S, Heiner J, Nagelhout J. Anesthesia case management for bariatric surgery. *AANA J*. 2011;79(2):147-60.
9. Ungern-Sternberg BS von, Regli A, Reber A, Schneider MC. Effect of obesity and thoracic epidural analgesia on perioperative spirometry. *Br J Anaesth*. 2005;94(1):121-7.

Cirrhosis with Portal Hypertension

Atul Prabhakar Kulkarni, Jigeeshu Vasistha Divatia, Sohan Lal Solanki

A 40-year-old male patient presents with a history of lump in the right hypochondrium. He received blood transfusion for a road trauma several years ago. He had a bout of jaundice 7 years ago and was found to be HBsAg positive. A CT scan now reveals cirrhosis of liver with a large nodule in the right lobe of the liver. He is posted for right hepatectomy.

Q. What are the functions of the liver?

Ans.

Metabolic Functions

Carbohydrate metabolism: Liver plays a vital role in both short- and long-term maintenance of glucose concentration within a narrow, normal range by *glycogenesis* (generation and storage of glycogen), *glycogenolysis* (glycogen breakdown), and *gluconeogenesis* (glucose synthesis).

Fat metabolism: Triglyceride oxidization for energy production, fatty acid broken down to acetoacetate for metabolism in other tissues. Liver also undertakes synthesis of lipoproteins, cholesterol, and phospholipids and conversion of carbohydrates and proteins into fatty acids and triglycerides.

Protein metabolism: This consists of deamination and transamination of amino acids, conversion of amino acids to glucose or lipids, synthesis of urea (removal of ammonia), nonessential amino acids, plasma proteins, and clotting factors.

Secretory and excretory functions: Liver secretes (400–800 mL/day) bile which contains bilirubin, water, cholesterol, electrolytes, bile acids, and phospholipids. Waste products are also secreted in bile. The bile acids (cholic and chenodeoxycholic) emulsify the fat particles in the gut-forming chylomicrons, which are important for absorption of fat-soluble vitamins such as vitamin K.

Vascular Functions

Phagocytic system: Sinusoids are lined by endothelial cells, interspersed with tissue macrophages (called Kupffer

cells). These remove particulate materials and microbes by phagocytosis and prevent their entry in systemic circulation.

Formation of lymph: Through fenestrations in sinusoidal endothelial cells, lymph is secreted in the “space of Disse” (space between hepatocytes and endothelium). From here it goes along the lymphatic capillaries associated with portal triads then to the systemic lymphatic system. Liver contributes 50% to total lymph production. If there is an increase in sinusoidal pressure (as in portal hypertension), lymph production is increased. Hence, we get ascites in portal hypertension.

Q. What is the blood supply to the liver? How is hepatic blood flow regulated?

Ans. The liver has dual blood supply—oxygenated blood from the hepatic artery (25%) and deoxygenated blood from the gut via the portal vein (75%). The liver is a very vascular organ and receives 30% of the resting cardiac output. It functions as a major vascular reservoir as it has the ability to store or release blood. The liver can compensate for moderate hemorrhage by ejecting adequate blood. In hypervolemic states, liver can act as a buffer by storing the blood.

Regulation of hepatic blood flow: Hepatic blood flow is regulated at three levels. First level is the systemic circulation. Changes in cardiac output, arterial resistance, and central venous pressure (CVP) will lead to changes in the hepatic blood flow. Regional macrocirculation is the second regulatory level. Hepatic blood flow depends on portal venous flow and resultant changes made by hepatic artery to buffer these alterations to maintain constant blood flow [autoregulation and hepatic arterial

buffer response (HABR)]. Hepatic microcirculation is the third level of regulation where changes occur in response to nitric oxide (NO) and endothelin concentration in the vessel walls.

Q. What are the liver function tests (LFTs)?

Ans. Liver function tests are done for detection, evaluation, and monitoring of liver damage. The commonly done tests include:

- *Serum enzymes:* Aminotransferases—alanine (ALT) and aspartate (AST): raised levels reflect hepatocellular injury
- *Cholestasis:* Alkaline phosphatase (ALP), 5'-nucleotidase, and γ -glutamyl transpeptidase (GGT)
- *Detoxification and excretory functions:* Serum bilirubin (total and direct), serum ammonia, and urine bilirubin
- *Biosynthetic function of the liver:* Serum albumin, serum globulin, and coagulation tests [prothrombin time (PT)].

Q. Is the albumin level or the PT a better test of acute liver dysfunction and why?

Ans. Prothrombin time collectively measures factors II, V, VII, and X. Biosynthesis of factors II, VII, IX, and X depends on vitamin K. Clotting factors have rapid turnover, with a half-life of 12 hours, as compared to albumin (whose half-life is 18–20 days with a slow 4% degradation/day). Therefore, measurement of the PT is the single best acute measure of hepatic synthetic function and helpful in both the diagnosis and assessing the prognosis of acute parenchymal liver disease.

Q. Which coagulation factors are synthesized in liver? Where are the other factors synthesized? Which factors are vitamin K-dependent factors?

Ans. All coagulation factors are made in the liver, except for von Willebrand factor (vWF) and factor VIII. vWF is produced in endothelium (in the Weibel–Palade bodies), megakaryocytes (α -granules of platelets), and subendothelial connective tissue. The vitamin K-dependent factors are:

- *Procoagulants:* Factors II, VII, IX, and X
- *Anticoagulants:* Proteins C, S, and Z.

Q. How does vitamin K enter the liver?

Ans. After ingestion, vitamin K gets emulsified by action of bile salts. Mixed micelles are then formed. These are taken up and formed into chylomicrons, which are secreted into the lacteals, within the intestinal villi, which drain into lymphatic vessels. These empty into the circulation via the thoracic duct. In muscles and adipose tissues, chylomicrons get converted to chylomicron remnants (containing vitamin K in a lipid core), which are then taken up by hepatocytes. Vitamin K is also formed in the body

by the action of gut bacteria (mainly bacteroides) and the main site of absorption in solution in the bile is the terminal ileum, vitamin K then reaches the liver by enterohepatic circulation. In the literature, however, there is doubt about the bioavailability of this vitamin K (produced by bacterial action) as it is tightly bound to the bacterial membrane. The largest amount is present in the colon (not terminal ileum), which does not have bile salts to make vitamin K soluble for absorption.

Q. Which conditions result in prolongation of the PT? Which conditions respond to vitamin K therapy?

Ans. Prothrombin time is prolonged in vitamin K deficiency, von Willebrand disease, and disseminated intravascular coagulation (DIC). Conditions which respond to vitamin K therapy are:

- Hemorrhagic disease of the newborn
- Fat malabsorption states such as cystic fibrosis, celiac disease, tropical sprue, Crohn's disease, ulcerative colitis, Ascaris infection, bacterial overgrowth, chronic pancreatitis, and short bowel syndrome
- Cholestasis due to common duct obstruction due to stones and strictures, primary biliary cirrhosis, cholangiocarcinoma, and chronic cholestasis (obstructive jaundice)
- *Drug interaction:* Cholestyramine (binds bile salts) causes fat malabsorption
- Vitamin K antagonists. Coumadin (Warfarin) inhibits vitamin K epoxide reductase and vitamin K reductase, and creates an intracellular deficiency. Cefamandole, cefoperazone, salicylates, hydantoins, rifampicin, isoniazid, and barbiturates are associated with vitamin K deficiency, but the mechanism is unknown
- Broad-spectrum antibiotics that destroy vitamin K-producing bacteria
- Diseases where coagulation inhibitors are produced: Lupus anticoagulant, antithrombins, and paraproteinemias, such as multiple myeloma.

Q. What is jaundice? How is bilirubin formed and how is it metabolized?

Ans. Jaundice is yellow discoloration of the skin, sclera (icterus), and other tissues caused by the deposition of excess circulating bilirubin.

Bilirubin formation and metabolism: Bilirubin is formed in the reticuloendothelial system, i.e., liver and spleen, when heme [70–80% from the breakdown of red blood cells (RBCs), and 20–30% derives from other heme proteins—prematurely destroyed erythroid cells in bone marrow and myoglobin and cytochromes in other tissues] is broken down. Unconjugated (indirect) bilirubin is water insoluble and is transported bound plasma albumin. Bilirubin is

taken up by the hepatocyte (without the albumin) by carrier-mediated membrane transport. In the presence of glucuronyltransferase, bilirubin is conjugated first to monoglucuronide and then to diglucuronide bilirubin (conjugated or water-soluble bilirubin). Conjugated bilirubin is secreted into the bile canaliculus. In the intestine, bacteria metabolize bilirubin to form urobilinogen. Most of this urobilinogen is converted to stercobilin, which is excreted in the stools. Some urobilinogen is excreted unchanged in urine. Some urobilinogen is reabsorbed, extracted, and excreted again by hepatocytes; this is called as enterohepatic circulation.

Q. What are the differences between hemolytic, hepatocellular, and obstructive jaundice?

Ans. *Hemolytic jaundice:* Hemolytic jaundice results from excessive breakdown of the RBCs. In the presence of normal liver function, serum bilirubin rises are modest and do not exceed 4 mg/dL. Other LFTs are normal with purely unconjugated hyperbilirubinemia.

Obstructive or cholestatic jaundice occurs when there is physical or physiological obstruction to bile flow. The obstruction may be intrahepatic or extrahepatic. Intrahepatic causes of obstructive jaundice are hepatitis, drug intoxication, primary biliary cirrhosis, cholestasis of pregnancy, metastatic cancer, and alcoholic liver disease. Extrahepatic causes are biliary calculus, stricture of common biliary duct, carcinoma of the common bile duct, carcinoma at the ampulla of Vater, pancreatitis or pancreatic pseudocyst, sclerosing cholangitis, and pancreatic cancer.

TABLE 1: Differences between hemolytic, obstructive, and hepatocellular jaundice.

Features	Hemolytic jaundice	Obstructive jaundice	Hepatocellular jaundice
Serum bilirubin	Modest rise, mostly unconjugated	High, mostly conjugated	High, both conjugated and unconjugated
Urine urobilinogen	High	Absent	Present (absent in predominantly cholestatic phase)
Urine bilirubin	Absent	Absent	High
Urine bile salts and pigments	Absent	Absent	Present
Stool stercobilin	High	Absent	Present
Liver enzymes	Normal	Alkaline phosphatase, GGT raised	Elevated transaminases
Coagulopathy	Absent	May be present	Present/Absent
Response to vitamin K	NA	Yes	Yes/No

This manifests as mixed hyperbilirubinemia. Some of the conjugated bilirubin reaches circulation and is excreted in urine, producing dark urine. Since most of the bilirubin does not reach the intestine, pale or white or clay colored stools are produced. Lack of bile salts leads to fat malabsorption and steatorrhea. Serum glutamic oxaloacetic transaminase (SGOT) and serum glutamic pyruvic transaminase (SGPT) are normal, and serum ALP is raised.

Hepatocellular jaundice is caused by hepatocyte damage, it is seen when >80% liver is damaged. It is seen with cirrhosis or viral hepatitis. There is increase in both unconjugated and conjugated bilirubin. The liver enzymes (SGOT and SGPT) are raised. **Table 1** summarizes the difference between the three types of jaundice.

Q. What are the types of viral hepatitis? Which is more likely to cause chronic liver disease and cirrhosis?

Ans. **Table 2** details the types of viral hepatitis (Hepatitis A through E) and their differences.

Hepatitis B and C both have chronic infection and carrier phases and cause cirrhosis. Hepatitis B [and hepatitis D virus (HDV)], when acquired during the neonatal period, can lead to cancer. Hepatitis C almost always leads to chronic hepatitis. Incidence of cirrhosis and hepatocellular carcinoma is very high with hepatitis C carriers.

Hepatitis "G" virus and "TT" virus, transfusion transmitted viruses, have been identified but they do not cause hepatitis.

Q. Describe the standard precautions in case of needlestick injury in a patient with hepatitis B or C.

Ans. Precautions developed for preventing exposure and transmission of disease in occupational setting, were previously called "universal precautions" implying precautions taken "everyone, everywhere, always." This has now changed to standard precautions meaning "standard of care." The components are:

- *Use of protective barriers.* Gloves for handling of mucus membranes, body fluids, broken skin, wear goggles, gowns, and masks during procedures.
- *Sharps* (needles, scalpels, suture material, bandages, and dressings), anything contaminated with any body fluid and waste: to be handled with gloves and disposed in designated puncture-resistant containers. While handling and disposing sharps, do not recap needles, put containers within arm's reach, use adequate light source when treating patients, and wear heavy-duty gloves when transporting sharps. Incinerate used needles to a sufficient temperature to melt.
- *Reusable instruments* such as speculums, surgical instruments, and thermometers must be thoroughly disinfected.

TABLE 2: Types and differences in hepatitis viruses.

Hepatitis	A virus	B virus	C virus (non-A–non-B)	D virus (delta body)	E virus
Spread	Feco-oral Contaminated food and water Oral/anal sexual contact	Blood and body fluid contact Sex Needles Mother to baby Human bite	Blood and body fluid contact Needles Mother to baby Sex (minimal)	Coinfection or superinfection with HBV	Feco-oral Contaminated food and water
Incubation	30 (15–50) days	60–90 (45–180) days	6–7 weeks (2–6 months)	60–90 (30–180) days	40 (14–60)
Symptoms of initial infection	Asymptomatic (especially HCV) jaundice, fever, myalgia, nausea, vomiting, anorexia, fatigue, dark urine, and pale bowel movements				
Chronic infection (infection for life)	No chronic disease	1–10% chronic can cause: Liver cell damage Cirrhosis Liver cancer	85% chronic can cause: Liver cell damage Cirrhosis Liver cancer	Common	No chronic disease
Prevention	Hepatitis A IgG up to 2 weeks after exposure	Hepatitis B IgG for PEP Recombinant vaccine	–	–	–
Treatment	Self-limiting	Interferon Lamivudine Adefovir PEGylated interferon Entecavir Telbivudine	PEGylated interferon plus ribavirin	Interferon?	None
Vaccine	Yes	Yes	No	HBV vaccine	No

(HBV: hepatitis B virus; HCV: hepatitis C virus; Ig: immunoglobulin; PEP: postexposure prophylaxis).

- *Immunizations* when available; take vaccines (hepatitis A and B)
- *Wound care after exposure:* Wash wounds gently (without vigorous scrubbing) with soap and water; let the wounds bleed freely. Irrigate mucosal surfaces with saline.

Q. Compare the infectivity of human immunodeficiency virus (HIV), hepatitis B, and hepatitis C. Discuss pre and postexposure prophylaxis (PEP) for these conditions.

Ans. Risk of transmission of infection depends on:

- No. of exposures to infected blood/fluids
- Prevalence of infected patients in the healthcare worker's area of practice
- Infectivity of the pathogen.

For the HIV, risk for transmission is not very high, the reported rates of seroconversion are as follows:

Percutaneous exposure to blood 0.3%, mucocutaneous exposure 0.03%, and exposure of intact skin 0%. The seroconversion rates for hepatitis B are 40%, while those for hepatitis C are 2%. However, with hepatitis C, 85% develop chronic hepatitis, 20% of these patients progress to cirrhosis, with 3% getting hepatocellular carcinoma (for vaccines, etc., **Table 2**).

For HIV, the commonly used drugs are: Zidovudine 250–300 mg BD, lamivudine 150 mg BD, and indinavir 800 mg TDS or efavirenz 600 mg OD. Depending on the type of exposure (**Flowchart 1**, 2 or 3 drugs), PEP has been shown to be effective in 81% patients. **Flowchart 1** summarizes

the evaluation of risk of HIV transmission and current recommendations for PEP.

Hepatitis B PEP: For adults, three (10 µg) injections of hepatitis B recombinant vaccine are given at 0, 1–2 months, and 5–6 months, respectively. A booster may be required after 5 years. Hepatitis B immunoglobulin (Ig) 500 IU at least needs to be given within 24–72 hours after exposure.

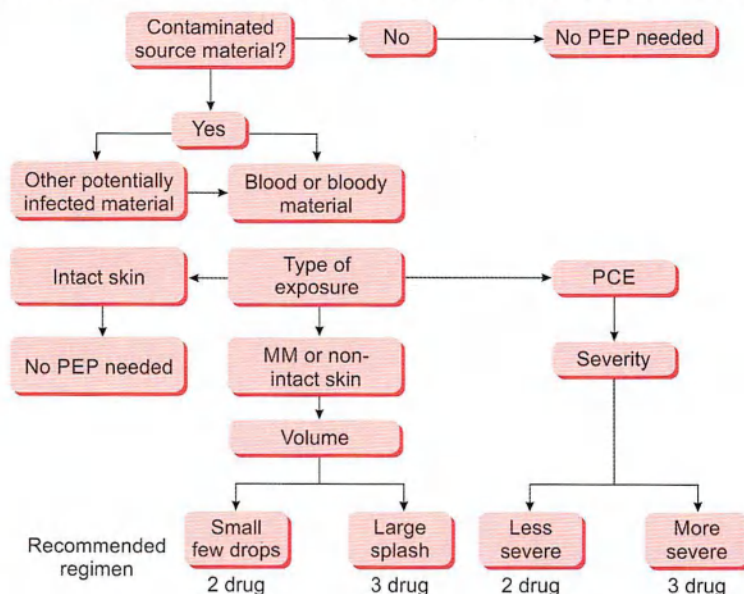
Q. What is cirrhosis of the liver?

Ans. Cirrhosis is a pathological condition where normal architecture of liver is distorted due to the development of fibrous tissue and the formation of regenerative nodules. This is accompanied by decreased hepatocellular mass as well as function and alteration of blood flow.

Q. What are the causes of cirrhosis?

Ans.

- Alcoholism
- Chronic viral hepatitis (hepatitis B and C)
- *Inherited metabolic liver diseases:*
 - Hemochromatosis
 - Wilson's disease
- Autoimmune hepatitis
- Nonalcoholic steatohepatitis
- *Biliary cirrhosis:*
 - Primary biliary cirrhosis
 - Primary sclerosing cholangitis
 - Autoimmune cholangiopathy.

Flowchart 1: Risk of human immunodeficiency virus transmission and currently recommended regimens.

(PEP: postexposure prophylaxis; PCE: primary care engagement).

TABLE 3: Adverse effects of alcohol.

System	Adverse effects
Central nervous system	Headache, cerebral atrophy, ataxia, and seizures Skull fractures, subdural hematoma
Psychiatry	Anxiety, panic, sedation, euphoria, irritability, restlessness, aggressiveness, violence, depression, sleep disturbances, memory and cognitive deficits, confabulation, hallucinations, and delusions
Eye	Blurred vision, loss of vision, or color vision abnormalities
Cardiovascular system	Cardiomyopathy, congestive heart failure, arrhythmias, coronary artery disease, hypertension, edema, and increased risk of hemorrhagic/ischemic stroke
Respiratory system	Pneumonia
Gastrointestinal system	Gastritis, ulcers, bleeding, malabsorption, diarrhea or constipation, and esophageal varices
Hepatopancreatic system	Fatty liver, hepatitis, jaundice, fibrosis, cirrhosis, coagulopathies, hypoproteinemia, ascites, and pancreatitis
Genitourinary system	Electrolyte imbalances, urinary tract infections, and sexual dysfunction
Peripheral nervous system	Paresthesias, peripheral neuropathies, extrapyramidal symptoms, and pain
Musculoskeletal system	Myalgias, cramps, atrophy, weakness, joint inflammation, worsening of rheumatoid arthritis, gout, bone ischemia, necrosis, and bone marrow depression
Hematological effects	Iron deficiency anemia, macrocytic anemia, leukopenia, and thrombocytopenia
Skin	Dermatitis, flushing, angiomas, urticaria, bruising, and sweating
Endocrine	Altered glucose tolerance, unstable diabetes, menstrual cycle irregularities, and gynecomastia

- Cardiac cirrhosis
- *Miscellaneous:*
 - Cystic fibrosis
 - Cryptogenic cirrhosis
 - α -1 antitrypsin deficiency.

Q. What are the adverse effects of alcohol?

Ans. Alcohol affects all organ systems in the body and produces a variety of adverse effects. The adverse effects also depend on the type of ingestion. A summary of adverse effects is given in **Table 3**.

Q. What are the complications of cirrhosis?

Ans. The complications of cirrhosis are:

- *Portal hypertension:*
 - Variceal bleeding, ascites, splenomegaly, and hypersplenism
- Liver cell failure
- Hepatic encephalopathy (HE)
- Hepatorenal syndrome (HRS)
- Spontaneous bacterial peritonitis (SBP)
- Hepatopulmonary syndrome (HPS)
- Portopulmonary hypertension (PPH).

Q. How is the portal vein formed? What is portal hypertension? What are the causes of portal hypertension?

Ans. Portal vein is formed by the superior mesenteric and splenic veins. Portal hypertension is defined as an increase in the hepatic venous pressure gradient to >5 mm Hg. It is caused by increased resistance to hepatic blood flow due to cirrhosis and regenerative nodules, and increased splanchnic blood flow due to splanchnic vasodilatation.

Causes of portal hypertension are:

- *Prehepatic causes:* Splenic arteriovenous (AV) fistula, splenic or portal vein thrombosis, and massive splenomegaly
- *Presinusoidal intrahepatic causes:* Sarcoidosis, schistosomiasis, nodular regenerative hyperplasia, congenital hepatic fibrosis, idiopathic portal fibrosis, early primary biliary cirrhosis, chronic active hepatitis, myeloproliferative disorders, and graft versus host disease
- *Sinusoidal intrahepatic causes:* Established cirrhosis and alcoholic hepatitis
- *Postsinusoidal intrahepatic causes:* Alcoholic terminal hyaline sclerosis and veno-occlusive disease
- *Postsinusoidal posthepatic causes:* Budd–Chiari syndrome, membranous inferior vena cava (IVC) web, right heart failure, and constrictive pericarditis.

Q. What are the complications of portal hypertension?

Ans. Variceal bleeding, splenomegaly, hypersplenism, and ascites are the main complications of portal hypertension.

Q. Which are the sites of portosystemic anastomoses?

Ans. The sites of portosystemic anastomosis are described in Table 4.

TABLE 4: Portosystemic anastomoses.

Site of portosystemic anastomosis	Portal component	Systemic component	Clinical effect
Lower esophagus	Esophageal branches of left gastric vein	Azygos veins	Submucosal gastroesophageal varices
Upper anal canal	Superior rectal vein	Middle inferior rectal veins	May be confused with hemorrhoids
Umbilical (anterior abdominal wall)	Veins of ligamentum teres	Superior/inferior epigastric veins	Caput medusae
Bare area of liver	Hepatic/portal veins	Inferior phrenic veins	
Patent ductus arteriosus (rare)	Left branch of portal vein	Inferior vena cava	
Retroperitoneal	Colonic veins	Body wall veins	Usually none

Q. How is acute variceal bleeding managed?

Ans. Management of acute variceal bleeding consists of:

- General resuscitation (fluid resuscitation and replacement blood and blood products, and hemodynamic and respiratory support)
- Specific measures to control the bleeding
- Measures to prevent HE.

The specific measures to control bleeding are:

- Pharmacological measures which cause vasoconstriction to stop bleeding. The drugs used are vasopressin [20 units in 200 mL over 20 minutes (check dose) with ECG (electrocardiogram) monitoring, terlipressin, glypressin, somatostatin, and octreotide (50–100 μ g/h as an infusion)].
- Mechanical measures inflatable balloons for tamponade by direct pressure. These are Sengstaken–Blakemore tube (esophageal and gastric balloon), Minnesota tube (modification of Sengstaken–Blakemore tube), and Linton–Nachlas tube with a single large gastric balloon.
- *Surgical measures:* Endoscopic variceal ligation.

Q. What is hepatorenal syndrome and how do you diagnose this?

Ans. Hepatorenal syndrome in simple language is the functional renal failure in the presence of hepatic failure. There are two types of HRS:

1. Type 1 HRS is a rapidly progressive renal failure that is defined by doubling of initial serum creatinine to a level >2.5 mg/dL or by 50% reduction in creatinine clearance to a level <20 mL/min in <2 -week period. There is always a precipitating factor identified for type 1 HRS.
2. Type 2 HRS is moderate, slowly progressive with a serum creatinine of >1.5 mg/dL. The type 2 HRS arises spontaneously and is the main underlying mechanism of refractory ascites.

There are major (mandatory) and minor (additional but not mandatory) criteria for the diagnosis of HRS.

Major criteria (2015) are as follows:

- Presence of cirrhosis with ascites
- Diagnosis of acute kidney injury (increase in sCr ≥ 0.3 mg/dL or 1.5 times over the baseline)
- No improvement of sCr after at least 2 days of diuretics withdrawal and volume expansion with albumin (1 g/kg of body weight per day up to a maximum of 100 g/day)
- Absence of shock
- No current or recent treatment with nephrotoxic drugs
- *No macroscopic signs of structural kidney injury:* Normal findings on renal ultrasonography, absence of proteinuria >500 mg/day, and absence of microhematuria.

Minor or additional criteria are as follows:

- Urinary volume < 0.5 L/day
- Urinary sodium < 10 mmol/L
- Urinary osmolality > plasma osmolality
- Urinary RBCs < 50/high-power field (HPF)
- Serum sodium < 130 mmol/L.

Q. What is hepatopulmonary syndrome and how do you diagnose this?

Ans. Hepatopulmonary syndrome is characteristic by a triad of the following:

- Arterial deoxygenation (widened PA-aO₂ gradient with or without hypoxemia)
- Intrapulmonary vasodilatation
- Liver disease.

Hepatopulmonary syndrome presents with a clinical feature of:

- Orthodeoxia
- Platypnea
- Dyspnea, digital clubbing, and cyanosis
- On X-ray of chest, there may be normal or increased interstitial pattern in the bases. Pulmonary function testing shows a low diffusion capacity and on arterial blood gas (ABG) testing shows varying levels of PaO₂ depending on the severity of HPS. HPS causes intrapulmonary vasodilatation that leads to arterial deoxygenation by V/Q (ventilation/perfusion) mismatch, intrapulmonary shunting, and limited oxygen diffusion. There is increase in pulmonary NO by endothelial and inducible NO synthase
- HPS can be diagnosed by microbubble contrast-enhanced transthoracic ECHO (echocardiography) and perfusion scanning with ^{99m}Tc-radiolabeled macroaggregated albumin (MAA) scan.

Q. What is portopulmonary hypertension and how do you diagnose this?

Ans. Pulmonary arterial hypertension is associated with portal hypertension, whether or not portal hypertension is secondary to an underlying liver disease and it is characterized by:

- Mean pulmonary artery pressure (PAP) >25 mm Hg at rest and >30 mm Hg with exercise
- Pulmonary artery occlusion pressure (PAOP) <15 mm Hg
- Pulmonary vascular resistance (PVR) > 240 dynes.s.cm⁻⁵.

Underlying pathogenesis for the development of PPH are imbalance of vascular mediators favoring vasoconstriction, endothelial damage with vascular remodeling due to excessive pulmonary blood flow, smooth muscle proliferation, and microvascular thrombosis.

Clinical features of PPH include:

- Fatigue, dyspnea, abdominal bloating, palpitations, chest pain or pressure, and syncope.
- Most common presenting symptom is dyspnea on exertion.
- Peripheral edema, elevation of the jugular venous pressure, loud P2, increased split of S2, pulsatile liver, or a right-sided S3 or S4.

Laboratory diagnosis is done by:

- *ECG*: It shows dilated right heart chambers, right axis deviation, and right bundle branch block
- *X-ray of chest*: It shows enlarged central pulmonary arteries and cardiomegaly
- *Pulmonary function tests*: Diffusion capacity may be decreased otherwise normal
- *Transthoracic Doppler ECHO*: Right ventricular systolic pressure (RVSP) > 40 mm Hg
- *Right heart catheterization*: Confirmatory.

Q. Describe a Sengstaken-Blakemore tube and the procedure. How is it inserted? Which balloon is inflated first and how? How is the Sengstaken-Blakemore tube fixed? Which balloon is inflated next and how? Which cuff is deflated and when? What if bleeding persists?

Ans. Sengstaken-Blakemore tube has three components: a gastric balloon, an esophageal balloon, and a gastric suction port. The balloons are for tamponade while the gastric port is for aspiration of blood from the stomach.

Procedure for insertion: Most patients will need to be intubated as it is a large tube and very uncomfortable for the patient; however, local anesthesia may be used. Patient is put in 45° head elevation. Alternatively, a left lateral decubitus position can be used. The balloons are checked by inflating and submerging under water for leaks, then ports are clamped after removing all air. Sometimes, a nasogastric tube is tied about 4 cm above the esophageal balloon and used for aspiration of blood from esophagus. The tube (and balloons) is lubricated and then it is passed (either nasally or orally) in the stomach up to the 50 cm mark. First, the gastric balloon is inflated with 100 mL increments and the pressure is measured. If pressure is above 15 mm Hg, the balloon may be in the esophagus, and it needs to be deflated and the tube is advanced further, till it is correctly positioned. Then, the gastric balloon is inflated with 450–500 mL of air, and the ports are clamped.

Proper placement can be confirmed by irrigating the gastric aspiration port with water while auscultating over the stomach. The tube is pulled back gently until resistance is felt. The tube is put on traction device (0.45–0.91 kg, generally a 0.5 L saline bottle) using a pulley.

Gastric port is aspirated to check for bleeding. If bleeding continues, the esophageal balloon is inflated to lowest pressure needed to stop bleeding (30–45 mm Hg) and then the port is clamped. A periodic watch is kept on the balloon pressure. If bleeding still continues, the traction is increased further to a maximum of 1.1 kg. Once bleeding is controlled, esophageal balloon pressure is decreased by 5 mm Hg every 3 hours to 25 mm Hg. This pressure is maintained for 12–24 hours. The esophageal balloon should be deflated for 5 minutes every 6 hours to help prevent esophageal necrosis. If bleeding recurs, the gastric balloon and, if necessary, the esophageal balloon may be reinflated for an additional 24 hours.

If bleeding persists beyond this time, alternative therapies need to be considered, these include endoscopic sclerotherapy and banding of the varices, transjugular intrahepatic portacaval shunt (TIPS), or surgery.

Q. Outline the management of portal hypertension in cirrhosis.

Ans. The primary treatment of portal hypertension is treatment of the cause, or removal of offending agents, if possible. Other measures include measures to lower portal venous pressure and treatment of complications:

- Nonselective β -blockers (such as propranolol) to reduce portal venous pressure.
- Low salt diet.
- *Diuretics for ascites:* Spironolactone to counteract sodium retention; loop diuretics can also be added.
- *Vasoactive drugs—reduce hepatic venous pressure gradient (in variceal bleeding):* Somatostatin or its analogs, octreotide and vapreotide, vasopressin or terlipressin-splanchnic vasoconstrictors but have ischemic side effects.
- *Portosystemic shunt procedures:*
 - *TIPS:* An intrahepatic shunt is created between portal and hepatic veins. This may lead to higher risk of HE. Frequently, stenosis occurs necessitating repeat procedure.
 - *Surgically created portosystemic shunts:* These are major procedures, not done routinely now, may be undertaken when TIPS is not possible.

Q. What are the types of surgical shunts performed for portal hypertension?

Ans. Three types of surgical shunts for portal hypertension have been described: total, partial, or selective shunts. With total shunts, portal venous flow is completely diverted away from liver, achieving total decompression. The examples are end-to-side and side-to-side portocaval shunt and

central splenorenal shunt. Partial shunts achieve partial decompression of the portal venous system and some portal flow is maintained, example is portocaval H-graft. The selective shunts also partly decompress portal circulation; the examples are distal splenorenal shunt and splenocaval shunt. The incidence of HE is high (~40%) with total shunts and drops (~10%) with partial and selective shunts. Surgical shunting is now rarely performed with the advent of TIPS procedure.

Q. What is hepatic encephalopathy? Discuss pathophysiology, signs of early encephalopathy, and grades of HE.

Ans. Hepatic encephalopathy or portosystemic encephalopathy is an alteration in the mental status and the cognitive function occurring in the presence of liver failure. HE occurs because the neurotoxins produced in the gut reach the brain due to portosystemic shunting as well as failure of liver to detoxify them due to reduced hepatic mass and function. The commonly cited reasons for HE include raised levels of ammonia, gamma aminobutyric acid (GABA) (due to altered GABA homeostatic mechanisms due to liver injury), and benzodiazepine receptor ligands [which may bind to GABA receptors and produce inhibitory central nervous system (CNS) effects]. In addition, ammonia may combine with alpha ketoglutarate in CNS forming glutamate which increases GABA synthesis.

Signs of HE: The first signs of HE can be subtle and nonspecific—change in sleep patterns, change in personality, irritability, and mental dullness. Later, confusion, disorientation, stupor, and eventually coma supervenes. HE is exceptional in that it has a fluctuating symptomatology which ranges from mild neurological impairment that may progress to deep coma fast and resolve again in hours.

Central nervous system examination: Trail-making test, where patient is asked to connect a series of 25 numbered circles as rapidly as possible using a pencil. A normal person can finish the test in 15–30 seconds; it is considerably delayed in patients with early HE. Micrographia may be an early sign. Patient may be asked to draw abstract objects or a fresh signature may be compared to previous signatures. More sophisticated testing includes electroencephalography and visual evoked potentials, which can detect mild forms of encephalopathy, but these are rarely clinically useful. **Table 5** lists grades of HE. Some articles divide Stage 4 further as follows:

- *Grade 4a:* Coma arousable by painful stimuli
- *Grade 4b:* Coma without response to painful stimuli.

TABLE 5: Grades of hepatic encephalopathy.

Grade	Level of consciousness	Intellectual function	Neurological findings	EEG
1	Lack of awareness Personality change Day/night reversal	Short attention	Incoordination Mild asterixis	Slowing (5–6 CPS) Triphasic
2	Lethargic Inappropriate behavior	Disoriented	Asterixis Abnormal reflexes	Slowing Triphasic
3	Asleep Rousable	Loss of meaningful communication	Asterixis Abnormal reflex	Slowing Triphasic
4	Unrousable	Absent	Decerebrate	Very slow (2–3 CPS), and delta

(EEG: electroencephalogram; CPS: complex partial seizures).

Q. How can HE be prevented? What is the mechanism of action of lactulose?

Ans. Mechanism of action of lactulose is not completely understood. One hypothesis is that on ingestion, lactulose reaches cecum unchanged, and is metabolized by enteric bacteria to lactate and acetate. This leads to a drop in the colonic pH. This acidification facilitates the passage of ammonia into the colon, where bacteria utilize ammonia as a metabolic substrate. This increases fecal nitrogen excretion and also reduces the amount of nitrogen reaching the portal blood with consequent reduction in serum ammonia level. Second hypothesis is that lactic acid production promotes growth of lactobacillus and other saccharolytic bacteria, which in turn suppresses proteolytic and urealytic bacteria. Third proposal is that lactulose serves as an energy source, which alters the metabolism of facultative urealytic and proteolytic bacteria, reducing ammonia production.

Q. What are the factors that precipitate HE?

Ans. Factors precipitating HE are given below:

- *Increased nitrogen load:*
 - Gastrointestinal (GI) bleeding
 - Excess dietary protein
 - Azotemia
 - Constipation
- *Disturbances of internal milieu:*
 - Hypovolemia
 - Metabolic alkalosis/acidosis
 - Hypoxia
 - Hyponatremia and hypokalemia
- *Drugs:*
 - Narcotics, tranquilizers, and sedatives
- *Miscellaneous:*
 - Infection
 - Surgery
 - Superimposed acute liver disease
 - Progressive liver disease
 - TIPS.

Q. What is ascites? What are the causes of ascites in cirrhosis?

Ans. Ascites is pathologic accumulation of fluid in the peritoneal cavity. Etiology of ascites is multifactorial.

Factors that cause generalized accumulation of fluid:

- Decreased synthetic function of liver leads to hypoalbuminemia, which leads to reduced oncotic pressure and leaking of fluid into the peritoneal cavity
- NO-induced vasodilation activation of rennin-angiotensin-aldosterone system with sodium and water retention.

Factors that lead to localized fluid accumulation in the abdominal cavity:

- Raised intrahepatic vascular resistance (with portal hypertension) along with splanchnic vasodilation leads to increased portal flow.
- Increased splanchnic production of lymph due to raised portal venous flow and due to seepage of lymph from lymphatic channels disrupted by nodular regeneration.

Q. What are the complications of ascites?

Ans. Complications of ascites include the development of refractory ascites and SBP.

Refractory ascites is defined as ascites unresponsive to 400 mg of spironolactone or 30 mg of amiloride plus up to 120 mg of furosemide daily for 2 weeks.

Treatment includes large-volume paracentesis (≤ 5 L) and peritoneovenous (LeVeen) shunt in selected patients with good hepatic reserve. Previous abdominal surgery, SBP, and large varices are relative contraindications to the shunt procedure.

Spontaneous bacterial peritonitis: This is a clinical syndrome, where ascites becomes infected in the absence of a recognizable cause of peritonitis. It is more common in patients with severely decompensated cirrhosis with jaundice. Diagnosis of SBP is by paracentesis. The “gold

standard" for diagnosis of SBP is a polymorphonuclear (PMN) count of >250 cells/ μL . Secondary bacterial peritonitis is an important differential diagnosis of SBP and shows the following features: (1) multiple organisms vs. single organism in SBP, (2) ascitic fluid protein concentration >1 g/dL (SBP <1 g/dL), (3) leukocytosis even with antibiotic therapy (SBP rapid fall in white cell count), and (4) culture remains positive (SBP becomes quickly sterile).

Q. How is ascites managed?

Ans.

- Salt-restricted diet (water restriction only if patient is hyponatremic)
- Spironolactone 100–200 mg/day (can be increased to 400–600 mg/day) and furosemide 40–80 mg/day (can be increased to 120–160 mg/day) might be added in patients with peripheral edema.
- *If ascites persists despite above refractory ascites:*
 - Repeated large volume abdominal paracentesis with albumin replacement as needed
 - TIPS.

Q. What are the clinical signs of liver cell failure?

Ans. The signs of hepatic failure include hair loss (alopecia), and flapping tremors of the body and tongue, gynecomastia, asterixis, parotid gland enlargement, digital clubbing, and muscle wasting. Fetor hepaticus is the slightly sweet, ammoniacal odor that may be seen in liver failure patients. Ascites, hydrothorax, prominent veins over the abdomen, and caput medusae (veins radiating from the umbilicus) are also seen. Widened pulse pressure and signs of a hyperdynamic circulation can occur in patients with cirrhosis as a result of fluid and sodium retention, increased cardiac output, and reduced peripheral resistance. Hyperpigmentation is seen with advanced chronic cholestatic diseases. Tendon xanthomata occur as a result of retention and high serum levels of lipids and cholesterol. A slate-gray pigmentation to the skin also occurs with hemochromatosis if iron levels are high for a prolonged period. Mucocutaneous vasculitis with palpable purpura, especially on the lower extremities, is typical of cryoglobulinemia of chronic hepatitis C but can also occur in chronic hepatitis B.

Spider angiomas and palmar erythema occur in both acute and chronic liver disease. Spider angiomas are superficial, tortuous arterioles, which typically fill from the center outward. Spider angiomas occur only on the arms, face, and upper torso; they can be pulsatile and may be difficult to detect in dark-skinned individuals. Testicular atrophy may also be seen (Fig. 1).

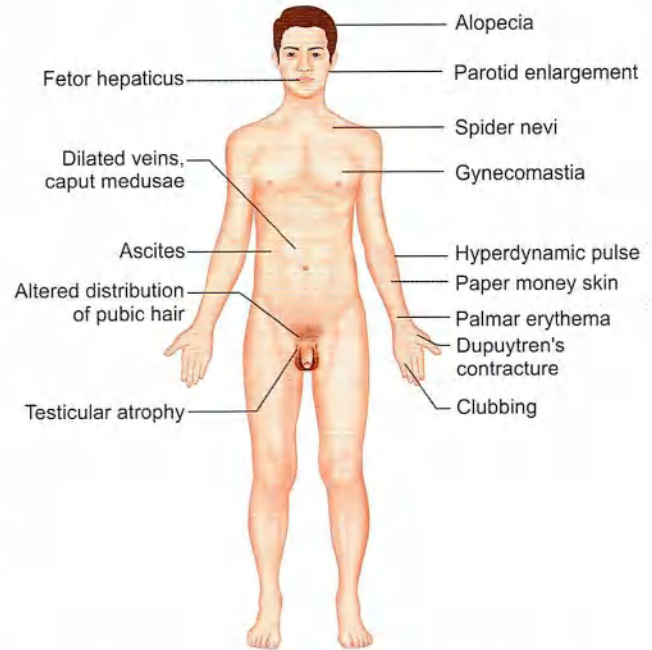


Fig. 1: Spider angiomas seen on different parts of the body.

Q. How is the risk assessment done in a case of chronic liver disease/cirrhosis? What is the Child-Pugh classification? What is MELD (model for end-stage liver disease), MELDNa, and PELD (pediatric end-stage liver disease model)?

Ans. There are various methods of staging of liver disease. Child-Turcotte-Pugh (CTP) classification (more commonly called as Child-Pugh classification) is a universally used method. The score ranges from 3 to 15. The grades are class A (score of 5–6), B (7–9), or C (>10). Class C indicates decompensated cirrhosis. Grade A represents “good” risk with 5% mortality, Grade B represents “moderate” risk with 10% mortality, Grade C represents “poor” risk with mortality $>50\%$ (Table 6).

Model for end-stage liver disease is another system to assess severity of liver disease, and was developed at Mayo Clinic to assess 3-month outcomes of patients undergoing TIPS. The calculation is done as follows (Table 6):

$\text{MELD} = 3.78 [\text{Ln serum bilirubin (mg/dL)}] + 11.2 [\text{Ln international normalized ratio (INR)}] + 9.57 [\text{Ln serum creatinine (mg/dL)}] + 6.43$. The maximum score is 40. The MELD-Na score includes serum sodium value to further refine the evaluation of the cirrhotic patient. The formula is $\text{MELD-Na} = [0.025 \times \text{MELD} \times (140 - \text{Na})] + 140$. MELD classification may be better at predicting 90-day mortality than Child-Pugh score even in patients undergoing other surgeries. A very recent study found that in patients undergoing nontransplant surgery, CTP predicted 30-day

mortality, MELD predicted 90-day mortality, and MELD-Na predicted 1-year mortality.

Pediatric end-stage liver disease score includes serum bilirubin, serum albumin, INR, age at entry into scoring, and growth failure. PELD score is used since 2002 to prioritize offer for pediatric liver transplant candidates and is an estimate of a candidate's risk of 3-month waiting list mortality.

TABLE 6: Child–Turcotte–Pugh classification of cirrhosis (score range 3–15).

Factor	1	2	3
Serum bilirubin	<2.0 mg/dL	2.0–3.0 mg/dL	>3.0 mg/dL
Serum albumin	>3.5 g/dL	3.0–3.5 g/dL	<3.0 g/dL
Prothrombin time Prolongation in seconds or INR	0–4 <1.7	4–6 1.7–2.3	>6 >2.3
Ascites	None	Easily controlled	Poorly controlled
Hepatic encephalopathy	None	Minimal	Advanced

(INR: international normalized ratio).

Q. Discuss in brief the management of HE.

Ans. Identify and correct the precipitating causes:

- Assess vital signs and volume status
- Evaluate for GI bleeding
- Eliminate sedatives or tranquilizers
- Screen for hypoxia, hypoglycemia, anemia, hypokalemia, metabolic alkalosis, and other potential metabolic or endocrine factors; correct as indicated.

Initiate ammonia-lowering therapy:

- **Dietary management:**
 - Previously protein restriction was advocated to reduce ammonia production
- Use nasogastric lavage, lactulose (30–45 mL syrup orally TDS/QID 300 mL retention enema until 2–4 bowel movements per day and mental status improvement) and/or other cathartics or enemas to remove source of ammonia from colon.
- Initiate treatment with lactulose or lactitol to produce 2–4 bowel movements per day.
- Consider oral nonabsorbable antibiotics to reduce intestinal bacterial counts:
 - Neomycin 4–12 g/day
 - Metronidazole and vancomycin have also been tried for this purpose.
- Consider treatment with flumazenil or another benzodiazepine-receptor antagonist.

Minimize potential complications of cirrhosis and depressed consciousness:

- Provide supportive care with attention to airway, hemodynamic, and metabolic statuses.

Q. What are the causes of anemia in a cirrhotic patient?

Ans. Cirrhotic patients may develop anemia due to chronic GI blood loss, nutritional deficiencies, or hypersplenism related to portal hypertension (causing early destruction of RBCs) and as a direct bone marrow suppression by alcohol on the Zieve's syndrome, a form of hemolytic anemia (with spur cells and acanthocytes) can occur in patients with severe alcoholic hepatitis. Erythropoietin though elevated as compared to normal patients are actually lower than those in patients with iron deficiency anemia, suggesting that the elevation is not enough to improve anemia.

Q. How is cirrhosis classified? What is decompensated cirrhosis?

Ans. Cirrhosis can be classified in a variety of ways:

Morphologic classification: Macronodular, micronodular, and mixed.

Histologic classification: Portal, postnecrotic, posthepatic, biliary, and congestive.

Etiologic classification: Genetic, toxic, infectious, biliary, vascular, and cryptogenic.

Functional classification: (Child–Pugh Classification) is the most commonly used classification. Class C indicates decompensated cirrhosis.

Q. What factors contribute to coagulopathy in cirrhosis?

Ans. The following factors contribute to coagulopathy in cirrhosis:

- Thrombocytopenia due to sequestration, decreased thrombopoietin, DIC, and qualitative platelet dysfunction
- Impaired coagulation factor synthesis and hypofibrinogenemia
- Coagulation factor consumption due to accelerated fibrinolysis and DIC
- Altered activity of coagulation factors due to γ carboxylation, dysfibrinogenemia, and vitamin K deficiency
- Decreased breakdown of plasminogen and increased fibrinolysis.

Q. What other investigations need to be performed in a patient with cirrhosis of liver?

Ans. Electrolytes: Electrolyte disturbances may be commonly seen in patients with peripheral edema and or ascites and

renal dysfunction. In absence of these, serum electrolytes are likely to be normal.

Renal function: Blood urea nitrogen (BUN) and urea. Renal dysfunction can occur in these patients due to imaging and treatment such as invasive diagnostic procedures, imaging studies (use of nephrotoxic contrast), nephrotoxic medications, urinary tract interventions with urinary tract infection (UTI) and obstruction, and therapies leading to volume depletion (diuretic therapy). IgA nephropathy is commonly seen with alcoholic liver disease. HRS is precipitated in these patients due to bacterial infection (SBP), GI bleeding and aggressive paracentesis, and drugs. Circulatory disturbances are also common in cirrhotics and these affect renal vasculature as well. In HRS, histology of kidney is often normal and renal dysfunction is reversed after liver transplant.

Cardiovascular system: ECG and echocardiography. Cardiac dysfunction is common in cirrhotic patients (called cirrhotic cardiomyopathy) and is distinct from alcoholic cardiomyopathy. The cardiac function appears normal at rest due to reduced peripheral resistance which allows cardiac output to be maintained but function decompensates in face of exercise or pharmacological stress. Proposed mechanisms for cirrhotic cardiomyopathy are sympathetic and parasympathetic autonomic dysfunction (due to either damaged peripheral nerves or changes in endogenous neurotransmitters) impaired β -adrenergic signal transduction. Decreased β -adrenergic receptor density and receptor desensitization have also been reported in cirrhotics. These patients have reduced sensitivity to catecholamines. Other problems in these patients include hypertension, cardiac failure, and arrhythmias [atrial fibrillation (AF), supraventricular tachycardia (SVT), and ventricular tachycardia (VT)].

Respiratory system: Cirrhosis can induce mild hypoxemia, atelectasis, and pleural effusion. In advanced cirrhotics, HPS (hepatic dysfunction, hypoxemia, and intrapulmonary vasodilatation) and PPH may occur.

Q. What changes in pharmacokinetics are expected in cirrhosis?

Ans. Volume of distribution: Cirrhotics are often hypervolemic and have a large volume of distribution. This means we have to give a larger initial dose of a drug to achieve an effect; however, because of hepatocellular dysfunction and reduced metabolism, the effect will be prolonged.

Protein composition and binding: Cirrhotics will often have lower levels of albumin; this means free fraction of

drugs, which are normally highly protein bound, will be higher. Presence of higher fraction of free drug (active drug), smaller doses will be needed for the clinical effect. Thiopentone is a classic example of this. When using thiopentone in cirrhotics, it is titrated to effect.

Biotransformation: Rate of metabolism of drugs will be slower, prolonging the half-life of drugs, this will be most apparent when top ups or infusions of drugs are given. Additionally, drugs with active metabolites will have even more prolonged action.

Q. What changes in anesthetic management do you expect in a patient with acute alcohol intoxication and a chronic alcoholic?

Ans. A patient with acute alcohol intoxication: Patients who present with acute intoxication for elective/emergency surgery are also likely to be chronic alcoholics, making it likely that all major organ systems are also likely to be affected (see above). Confusion, aggression, and CNS depression presenting as somnolence or even coma may occur. Obtaining consent is a problem as they have to be considered as incapable of providing informed consent till the effect of alcohol wears off. Metabolic derangements include hypoglycemia and even alcoholic ketosis. Sedative drugs are less likely to be needed. They are to be treated as having full stomach, due to delayed gastric emptying, thus rapid sequence induction is needed. Alcohol-induced depression means hypotension is likely after induction. One may find metabolic conditions such as hypoglycemia and alcoholic ketoacidosis. Excessive dextrose may precipitate Wernicke's encephalopathy. It is caused by thiamine (vitamin B₁) deficiency and is characterized by the classic triad of ataxia, ophthalmoplegia, and acute mental confusion. Treatment includes oral or intramuscular thiamine administration (100 mg OD for 3 days) and volume restoration.

A chronic alcoholic: A chronic alcoholic is likely to have disorders of all major systems (see adverse effects of alcohol). Volatile anesthetics and induction agent's dose requirement will be higher. The initial dose requirement of most drugs will be higher (increased volume of distribution) but their effect is prolonged. Thrombocytopenia, leukopenia, anemia, coagulopathy, and electrolyte derangements (hypomagnesemia, hypophosphatemia, and hypocalcemia) are common. Postoperative course may be complicated by alcohol withdrawal syndrome (AWS). This may present as tremor, gastric upset, sweating, hypertension, hyperreflexia, anxiety, and agitation may worsen to delirium, hallucinations, and seizures, within 6–24 hours (up to 5 days) without alcohol.

Q. If the patient has to undergo a lower abdominal surgery or a hernia, which methods would you prefer—regional/spinal/epidural/GA? Why?

Ans. The most important consideration in a cirrhotic patient undergoing anesthesia and surgery is the maintenance of hepatic blood flow and oxygenation. A decrease in hepatic blood flow due to reduced HABR and simultaneous drop in cardiac output will lead to reduced hepatic and renal blood flow as well, making the patient susceptible to the development of hepatic decompensation and renal failure.

Herniorrhaphy can be successfully carried out under inguinal block. For lower abdominal surgery, a central neuraxial blockade can be safely undertaken provided coagulopathy is tackled properly by platelet transfusion, fresh frozen plasma (FFP), and cryoprecipitate administration. INR should be maintained below 1.4 (PT within 4 seconds of control), at the time of epidural catheter insertion and removal. Other concern is hypotension and reduced hepatic blood flow which can be managed with fluid infusion (optimizing preload) and administration of vasopressors to keep the blood pressure normal.

Q. What anesthesia technique would you use for this patient undergoing hepatic resection?

Ans. A combination of GA and epidural anesthesia is best for this patient. The important consideration is to maintain the hepatic blood flow within normal limits by ensuring normal blood pressure.

Q. Which agents are preferred for premedication?

Ans. In absence of HE and moderate hepatic dysfunction, small dose of benzodiazepine (midazolam) can be safely used. In patients with even early HE, both benzodiazepines and opiates should be avoided.

Q. How are benzodiazepines metabolized? Which of these do not have active metabolites? What is the half-life of metabolites of diazepam and midazolam?

Ans. All benzodiazepines are metabolized in the liver in two phases, oxidation followed by conjugation. Lorazepam, oxazepam, and temazepam do not have active metabolites. Diazepam is metabolized to nordiazepam (60 hours), oxazepam (8 hours), and temazepam (12 hours). Midazolam is metabolized to α -hydroxymidazolam, an active metabolite with a short half-life and it does not contribute to prolongation of action of midazolam.

Q. Which induction agents are preferred? What dose adjustments may be required?

Ans. There are no preferred induction agents. Thiopentone can be used safely provided one understands that the dose needs to be reduced (in view of increased free fraction of drug due to reduced albumin levels) and titrated to sleep.

Propofol has been safely used as an induction agent and as continuous infusion (for 8 hours) for maintenance of anesthesia. It was observed that though the volume of distribution was significantly increased and total body clearance decreased in cirrhotic patients, this did not increase terminal elimination half-life. Cirrhotic patients with moderate hepatic dysfunction still metabolize etomidate normally.

Q. What is the impact of liver disease on pseudocholinesterase production and duration of action of succinylcholine?

Ans. In cirrhotic patients with hepatic dysfunction, the levels of pseudocholinesterase will be decreased. However, the duration of action of succinylcholine is not prolonged till pseudocholinesterase levels drop to <50% of normal.

Q. What changes in the dose and dose intervals of nondepolarizing muscle relaxants are required?

Ans. In patients with cirrhosis, the initial dose requirement of nondepolarizing muscle relaxants will be increased (due to increased volume of distribution); however, smaller subsequent doses will be needed as duration of action of most relaxants (except atracurium) is prolonged. The best way to use a muscle relaxant is to use neuromuscular monitoring.

Q. Which muscle relaxants are preferred? Enumerate protein-binding, alternate route of excretion/metabolism for all common muscle relaxants. Are there any concerns while using atracurium or vecuronium?

Ans. Atracurium and cisatracurium are the preferred muscle relaxants. Atracurium is mainly metabolized by Hoffmann elimination (spontaneous degradation at physiological pH and temperature) into laudanosine and monoacrylate. Smaller amount is hydrolyzed by nonspecific plasma esterases. Since no organ is required for Hoffmann elimination, duration of action is not prolonged with either renal or hepatic dysfunction. Laudanosine is eliminated by hepatic clearance and it will accumulate in presence of liver function, the concern being seizures (also accumulates in patients with renal failure). However, doses of atracurium during anesthesia are too small to result in the levels of laudanosine that cause seizures.

Pancuronium is about 70–90% protein-bound, mainly metabolized by the liver to 3-hydroxy (50% as potent), 17-hydroxy metabolite, and 3,17-dihydroxy metabolites (both less active). About 40% is excreted unchanged by kidney. About 10% is excreted in bile. In abnormal hepatic function, the volume of distribution is increased; however, more free drug is available due to reduced plasma albumin that can increase renal elimination. Duration of action

is prolonged. About 60–80% of vecuronium bromide is bound to plasma protein.

Vecuronium, in the liver, is deacetylated to the active metabolites 3-hydroxy (approximately 50% potent) and 17-hydroxy, and 3,17-dihydroxyvecuronium. About 25–50% of total dose is excreted in bile in up to 42 hours. About 60–80% is bound to protein. Remaining drug and metabolites are excreted via kidney. Studies in patients with hepatic dysfunction and cholestasis shown that after larger doses (0.2 mg/kg), the duration of neuromuscular blockade may be considerably prolonged.

Rocuronium is 30% protein bound. It deacetylated in the liver to 17-desacetyl-rocuronium (1/20 as active) and undergoes hepatobiliary excretion. Studies in patients with cirrhosis have shown an increased initial dose requirement (increased volume of distribution) almost doubled duration of neuromuscular blockade (reduced clearance).

Q. How are morphine and other opiates metabolized?

Ans. Morphine is largely metabolized in liver while smaller amounts are metabolized in kidney and brain. Morphine-3 glucuronide (M3G) and morphine-6-glucuronide (M6G) are the major and active metabolites of morphine, and in patients with hepatic dysfunction, the duration of action of morphine is therefore prolonged. The glucuronides are then eliminated via bile and urine. In the cirrhotics with abnormal liver function, morphine plasma clearance is significantly lower; the terminal elimination half-life is longer.

Pethidine is hydrolyzed to pethidinic acid and demethylated to norpethidine in the liver. Norpethidine is an active metabolite with a longer elimination half-life (8–12 hours). It is structurally similar to atropine and has anticholinergic activity leading to convulsant and hallucinogenic effects, on cumulation in patients with renal failure. Pethidine metabolites are conjugated with glucuronic acid and excreted into the urine.

Fentanyl is also metabolized in liver to norfentanyl by oxidation. Remifentanyl is metabolized in the plasma by nonspecific tissue and plasma esterases.

Q. How do changes in liver blood flow and liver function affect the metabolism of drugs by the liver? What are the effects of surgical manipulations and regional anesthesia?

Ans. Hepatic drug clearance (metabolism) depends on two factors: the hepatic blood flow (the greater the flow, the greater the drug presented for clearance) and intrinsic enzyme metabolizing hepatic capacity for that drug. Hepatic extraction ratio is the amount of free drug taken up by the liver during one passage. When the extraction ratio is 1, all the drug reaching the liver (100% of drug amount) is

taken up by the liver, and cleared from the blood. Thus, the blood leaving the liver shall have very small amount left in it. Such drugs are called *high intrinsic clearance drugs*. The examples are propranolol, lignocaine, and pethidine. In case of high clearance drugs, the elimination is then directly proportional to the hepatic blood flow. Drugs with low intrinsic clearance are not as dependent on the hepatic blood flow, as higher blood flow will not increase their elimination further, the examples being diazepam, phenytoin, and coumadin.

Anesthesia leads to reduction in hepatic blood flow due to a reduction in blood pressure and cardiac output. Inhalational agents have a greater effect on the hepatic blood flow than the intravenous agents. Halothane causes the greatest reduction, then isoflurane, desflurane, and sevoflurane show decreasing effects. Intravenous anesthetic agents, such as propofol, have no or very minimal effect on HABR (an intrinsic regulatory mechanism to maintain total hepatic blood flow with changes in portal perfusion pressure—see above).

Upper abdominal surgical manipulations can reduce the hepatic blood flow by over 60% due to splanchnic vasoconstriction. Subarachnoid block to T4 level will reduce the blood flow by 20%.

Q. Which inhalation agents can be used? Are all fluorinated volatile anesthetics safe? Can halothane be used? What is halothane hepatitis?

Pathophysiology of halothane hepatitis (National Halothane Commission).

Ans. Isoflurane is the safest agent, i.e., with the least reduction in hepatic blood flow and with minimal (0.2%) being converted to acetylated protein adducts. Halothane hepatitis is hepatotoxicity associated with halothane administration. Two types of hepatotoxicity are seen with halothane, type I is common, benign, and self-limiting and occurs due to reductive biotransformation of halothane. A transient mild increase in serum transaminase and glutathione S-transferase concentrations is seen. Type I hepatotoxicity is not characterized by jaundice or clinically evident hepatocellular disease. Halothane hepatitis (type II hepatotoxicity) is probably immune mediated. Oxidative metabolism of halothane leads to the formation of trifluoroacetyl metabolites, which bind to liver proteins. In genetically susceptible individuals, antibodies are generated against these complexes and hepatotoxicity results. This is characterized by fever, jaundice, very high serum transaminase levels, and pathologically by massive centrilobular liver necrosis leading to fulminant liver failure, with a high (50%) mortality rate. Type II hepatotoxicity appears to be immune mediated. The National Halothane Study (1959–1962) was a retrospective study of possible

relation between postoperative mortality due to hepatic necrosis due to the use of halothane, and found that the risk of fatal hepatotoxicity was 1 in 35,000.

Q. What is pharmacological preconditioning? Does it work?

Ans. Prophylactic administration of sevoflurane and isoflurane has been used in cardiac surgery to prevent cardiac dysfunction and reduce microscopic abnormalities due to reperfusion after an ischemic insult in patients undergoing cardiac surgery. A 30-minute administration of sevoflurane before portal clamping was tried as a strategy for hepatic protection. The authors found that postoperative enzyme elevation was much less in patients who received sevoflurane as compared to those who did not. Another study in patients undergoing hepatic transplant found that sevoflurane preconditioning improved early allograft function. This has yet to become a standard of care in all centers as more studies are awaited.

Q. What are the mechanisms of drug hepatotoxicity? Which drugs are hepatotoxic?

Ans.

- *Mechanisms of drug hepatotoxicity:* Classically, these are of two types, very often it is difficult to ascribe a particular mechanism, and often these may overlap.
- *Intrinsic or predictable drug reactions:* The drug or its metabolite causes reproducible injury across species and is dose dependent. Acetaminophen and carbon tetrachloride are examples of this type of drugs.
- *Idiosyncratic drug reactions:* Idiosyncratic reaction is an adverse reaction that does not occur in most patients at normal dose of a drug and is not like the known effect of the drug. This is of two types: hypersensitivity or immune-allergic and metabolic-idiosyncratic. When hepatotoxicity is seen with fever, rash, eosinophilia, and antidrug antibodies, and recurs swiftly on repeat exposure, it is said to be an immune-mediated reaction. In absence of these features, it is likely to be metabolic idiosyncrasy:
 - *Hypersensitivity:* The Hapten hypothesis proposes that this reaction occurs not due to the drug itself, but by irreversible binding of the drug to a protein. This complex then acts as an allergen and initiates the reaction. Phenytoin and penicillins are drugs which cause hypersensitivity reaction.
 - *Metabolic-idiosyncratic:* It occurs due to an indirect metabolite of the drug with variable and often delayed response. Unlike intrinsic hepatotoxins, the response rate is variable and can occur within a week or up to 1 year later. Examples are ketoconazole and halothane.

Q. What intraoperative monitoring will you use?

Ans. Noninvasive monitoring will consist of ECG, noninvasive blood pressure (NIBP), end-tidal CO₂ (ETCO₂), pulse oximetry, fractional inspired concentration of volatile agent (optional), peripheral nerve stimulator for neuromuscular blockade monitoring, and temperature monitoring. Invasive monitoring is extremely important in this patient. Invasive arterial pressure monitoring is extremely useful, as there are considerable fluctuations in blood pressure. This is accentuated during dissection around the IVC. Arterial line is also useful for ABG sampling to assess for metabolic disturbance, hypoxemia, and hypercarbia. Intraoperative bleeding has been shown to be considerably reduced when the CVP is maintained at CVP <5 mm Hg, so a central venous catheter is very useful in these patients. Intermittent blood glucose monitoring and hourly urine output should also be monitored. Coagulation monitoring either with thrombocyte count and PT or thromboelastography is also necessary.

Q. How will you manage fluid therapy in the intraoperative period?

Ans. Recently, individualized goal-directed fluid therapy (GDFT) based on patient's individual fluid responsiveness and real time monitoring of a particular hemodynamic goal is being used. This is based on physiological principle of the Frank-Starling curve. GDFT utilizes a cardiac output monitor to individualize fluid therapy. This technique is also called as stroke volume (SV) optimization technique, as the goal is to optimize the patient's SV throughout the perioperative period. Different hemodynamic goals to direct the fluid therapy have been suggested, for example, pulse pressure variation, SV variation, or lactate.

Pulse pressure variation or PPV [(maximum pulse pressure – minimum pulse pressure)/mean pulse pressure] can be calculated if an arterial line is in situ. PPV >10% suggests that the patient is fluid responsive and indicates that SV is sensitive to fluctuations in preload caused by the respiratory cycle. PPV-guided fluid therapy is a better way to give fluids in these cases.

Q. What are the potential problems and what precautions would you take during a prolonged surgery?

Ans. Potential problems during prolonged surgery in a cirrhotic patient include hypovolemia, hypotension, and hypoxemia secondary to hemorrhage. Hemorrhage occurs because of coagulopathy (if not completely corrected preoperatively and hypothermia) cirrhosis itself, and if the CVP is not maintained at <5 mm Hg. Corrective measures aim at FFP, cryoprecipitate, and platelet replacement. Adequate blood loss replacement with packed RBCs is also necessary.

Hypothermia frequently occurs due to convective loss of heat from the open abdomen. Active warming measures should be instituted including warming blankets and infusion of warm intravenous fluids.

Q. How will you manage excessive bleeding?

Ans. Excessive bleeding should be managed by correcting coagulopathies, active warming, and adequate replacement with blood and blood products. As mentioned earlier, reducing the CVP will also help reduce bleeding.

Q. How will you manage postoperative analgesia? What precautions should be taken if epidural analgesia is used?

Ans. In our patient, the preoperatively placed epidural catheter can be continued to be used for providing postoperative analgesia. Dilute (0.0625%) solutions of bupivacaine can be used as an infusion. Small doses of acetaminophen 325 mg in 3–4 doses/day can be added. Acetaminophen in these doses is shown to be safe in patients with reasonably preserved hepatic function. Nonsteroidal anti-inflammatory drugs (NSAIDs) such as diclofenac may also be added for a synergistic effect. The catheter should be removed once the INR normalizes, which usually takes 4–6 days. If earlier removal is contemplated for some reason, then catheter should be removed after infusing FFP to correct INR.

Q. What are the causes of postoperative jaundice?

Ans. Postoperative jaundice can be multifactorial in origin. The causes are as follows:

- Bilirubin overproduction
- *Hepatocellular necrosis:*
 - Ischemic liver injury
 - Viral hepatitis
 - Drug-induced hepatitis
 - Hepatocellular necrosis after liver transplantation
- *Intrahepatic cholestasis:*
 - Benign postoperative cholestasis
 - Infection
 - Medications
 - Intrahepatic cholestasis after liver transplantation
- *Extrahepatic cholestasis:*
 - Upper abdominal surgery
 - Acalculous cholecystitis
 - Extrahepatic biliary obstruction after liver transplantation

• *Miscellaneous causes:*

- Genetic disorders of bilirubin metabolism
- Total parenteral nutrition.

SUGGESTED READING

1. Beck-Schimmer B, Breitenstein S, Urech S, De Conno E, Wittlinger M, Puhon M, et al. A randomized controlled trial on pharmacological preconditioning in liver surgery using a volatile anesthetic. *Ann Surg.* 2008;248(6):909-18.
2. Bosch J, Reverter JC. The coagulopathy of cirrhosis: myth or reality? *Hepatology.* 2005;41(3):434-5.
3. Causey MW, Steele SR, Farris Z, Lyle DS, Beitler AL. An assessment of different scoring systems in cirrhotic patients undergoing nontransplant surgery. *Am J Surg.* 2012;203(5):589-93.
4. Hoetzel A, Ryan H, Schmidt R. Anesthetic considerations for the patient with liver disease. *Curr Opin Anaesthesiol.* 2012;25(3):340-7.
5. Huffmyer JL, Nemergut EC. Respiratory dysfunction and pulmonary disease in cirrhosis and other hepatic disorders. *Respir Care.* 2007;52(8):1030-6.
6. Jiménez W, Arroyo V. Origins of cardiac dysfunction in cirrhosis. *Gut.* 2003;52(10):1392-4.
7. Kisor DF, Schmith VD, Wargin WA, Lien CA, Ornstein E, Cook DR. Importance of the organ-independent elimination of cisatracurium. *Anesth Analg.* 1996;83(5):1065-71.
8. Ko JS, Gwak MS, Choi SJ, Kim GS, Kim JA, Yang M, et al. The effects of desflurane and propofol-remifentanyl on postoperative hepatic and renal functions after right hepatectomy in liver donors. *Liver Transpl.* 2008;14(8):1150-8.
9. Martin P. Perioperative considerations for patients with liver disease. *Cleve Clin J Med.* 2009;76(Suppl 4):S93-7.
10. Minou AF, Dzyadzko AM, Shcherba AE, Rummo OO. The influence of pharmacological preconditioning with sevoflurane on incidence of early allograft dysfunction in liver transplant recipients. *Anesthesiol Res Pract.* 2012;2012:930487.
11. Tyma A, Theodoraki K, Tsaroucha A, Arkadopoulos N, Vassiliou I, Smyrniotis V. Anesthetic considerations in hepatectomies under hepatic vascular control. *HPB Surg.* 2012;2012:720754.
12. Van Miert MM, Eastwood NB, Boyd AH, Parker CJ, Hunter JM. The pharmacokinetics and pharmacodynamics of rocuronium in patients with hepatic cirrhosis. *Br J Clin Pharmacol.* 1997;44(2):139-44.
13. World Health Organization. (2007). Post-exposure prophylaxis to prevent HIV infection: Joint WHO/ILO guidelines on post-exposure prophylaxis. (PEP) to prevent HIV infection. [online] Available from: <http://www.who.int/hiv/pub/guidelines/PEP/en/> [Last accessed February 2020].
14. Yeung E, Yong E, Wong F. Renal dysfunction in cirrhosis: diagnosis, treatment and prevention. *Medscape Gen Med.* 2004;6(4):9.

Laparoscopic and Robotic Cholecystectomy

Priya Ranganathan, Debashree Lahiri

A 50-year-old female patient presents with the history of jaundice 3 months ago, and pain in the right hypochondrium with anorexia and nausea since 1 month. She is diagnosed to have gallstones and is posted for a laparoscopic cholecystectomy.

Q. What is laparoscopy?

Ans. Laparoscopy is the visualization of the abdominal cavity through an endoscope.

Q. What are the advantages of laparoscopic surgery over open surgery?

Ans.

- Reduced stress response to surgery
- Rapid return of gastrointestinal (GI) function
- Reduced postoperative pain and analgesic requirements
- Improved postoperative respiratory function
- Reduced recovery time
- Improved cosmetic appearance
- Less postoperative wound infection.

Q. Describe the technique of laparoscopy.

Ans. Laparoscopic surgery involves insufflation of a gas (usually carbon dioxide) into the peritoneal cavity under pressure to separate the organs from the abdominal wall. This produces a pneumoperitoneum, which causes an increase in intra-abdominal pressure (IAP). Gas is insufflated into the peritoneal cavity at an initial rate of 4–6 L/min which gets cut off when the IAP exceeds the pressure limits set by the clinician (usually 12–14 mm Hg). After that the pneumoperitoneum is maintained by a constant gas flow of 200–400 mL/min.

Q. What should be the properties of the gas used for insufflation?

Ans. The following properties are desirable in the gas used for insufflation:

- It should not cause significant physiological changes
- Minimal absorption from the peritoneal cavity
- If absorbed, it should be rapidly excreted

- Should not support combustion
- Should be highly soluble in blood to minimize risk of gas embolism.

Q. Enumerate the advantages and disadvantages of various gases which may be used for insufflation.

Ans.

Table 1 shows the advantages and disadvantages of the various gases.

TABLE 1: Advantages and disadvantages of various gases.

Gas	Advantages	Disadvantages
Air, oxygen	• Easily available • Inexpensive • Limited physiological effects	Support combustion
Nitrogen	• Does not support combustion • Poorly absorbed—avoids hypercapnia	Low blood solubility—dangerous consequences if gas embolization occurs
Nitrous oxide	May be beneficial in patients undergoing procedures under regional anesthesia	Supports combustion
Helium	• Poorly absorbed—avoids hypercapnia • Does not support combustion	• Low blood solubility—dangerous consequences if gas embolization occurs • Not cost-effective
Argon	• Poorly absorbed—avoids hypercapnia • Does not support combustion	• Low blood solubility—dangerous consequences if gas embolization occurs • Not cost-effective • Decrease in hepatic blood flow

Contd...

Contd...

Gas	Advantages	Disadvantages
Carbon dioxide	• Does not support combustion • High blood solubility • Inexpensive • Cleared rapidly in postoperative period	Absorbed in large quantities from the peritoneal cavity with significant physiological effects

Q. Describe the ventilatory and respiratory changes during laparoscopy with CO₂ pneumoperitoneum.

Ans.

Effect of Raised Intra-abdominal Pressure

The increase in IAP results in cephalad displacement of the diaphragm, reducing functional residual capacity (FRC) and compliance by as much as 30–50%. Peak airway pressure and plateau pressures rise by 50 and 80%, respectively, and pulmonary compliance is reduced by 47%. This results in an increase in the airway resistance and work of breathing. Hypoxemia may result from atelectasis and intrapulmonary shunting, and this is more common in obese patients and those with underlying cardiopulmonary disease.

Increase in Partial Pressure of CO₂

CO₂-pneumoperitoneum results in a progressive increase in PaCO₂ which reaches a plateau 20–30 minutes after the beginning of intraperitoneal insufflations. The increase in PaCO₂ depends on the IAP and ranges between 15 and 30%. In patients without cardiorespiratory disease, the increased PaCO₂ results mainly from CO₂ absorption from pneumoperitoneum. It does not significantly modify either physiologic dead space or shunt. As a consequence, the gradient between PaCO₂ and end-tidal PCO₂ (ETCO₂) does not change and ETCO₂ monitoring adequately reflects PaCO₂ and can be used to guide adjustment of ventilation to prevent hypercapnia. In patients with cardiorespiratory disease or in cases of acute cardiopulmonary disturbances, pneumoperitoneum impairs pulmonary ventilation and perfusion, resulting in increased physiologic dead space, reduced alveolar ventilation and increased PaCO₂ to ETCO₂ gradient. This leads to a greater increase in PaCO₂ and more hyperventilation is required to prevent hypercapnia. In these patients, capnometry no longer provides reliable monitoring of PaCO₂. Carbon dioxide diffuses to the body more during extraperitoneal than intraperitoneal insufflations. Also extraperitoneal carbon dioxide insufflation leads to higher PaCO₂ values in the postoperative period.

Change in Minute Ventilation

During laparoscopy with local anesthesia, minute ventilation increases by as much as 60% to maintain normocapnea. During general anesthesia with spontaneous

breathing, the compensatory hyperventilation is insufficient to avoid hypercapnia because of anesthetic-induced respiratory depression. In patients on controlled ventilation, a 15–20% increase in minute ventilation is needed to prevent hypercapnia.

Q. What respiratory complications can occur during laparoscopy?

Ans. The major complications which can occur during laparoscopy are:

- Carbon dioxide subcutaneous emphysema—due to extraperitoneal insufflation
- Pneumothorax, pneumomediastinum, and pneumopericardium
- Endobronchial intubation—due to cephalad movement of the diaphragm
- Gas embolism
- Aspiration of gastric contents.

Q. Describe the hemodynamic changes during laparoscopy with CO₂ pneumoperitoneum.

Ans.

Effects of Hypercapnia

Hypercapnia due to CO₂ pneumoperitoneum activates the sympathetic nervous system leading to an increase in blood pressure, heart rate, myocardial contractility, and arrhythmias. It also sensitizes the myocardium to catecholamines, particularly when volatile anesthetic agents are used.

Effects of Raised Intra-abdominal Pressure

At IAP levels <15 mm Hg, venous return is augmented as blood is squeezed out of the splanchnic venous bed, producing an increase in cardiac output.

At IAP levels >15 mm Hg, cardiac output decreases transiently due to:

- *Decreased venous return:*
 - Compression of the inferior vena cava along with the surrounding collateral vessels
 - Pooling of blood in the legs
 - Increased venous resistance.
- Myocardial depressant effect of anesthesia induction agents
- *Raised systemic vascular resistance:*
 - Release of neurohumoral factors
 - Increased vascular resistance of organs.

The increase in systemic vascular resistance causes an increase in arterial pressures. Right atrial and pulmonary artery occlusion pressures are elevated due to increased intrathoracic pressures and these cannot be considered to be reliable indices of cardiac filling pressure during pneumoperitoneum. Cardiac arrhythmias during laparoscopy are due to multiple

causes—hypercapnia, peritoneal stimulation, reduced venous return, hypovolemia, and venous gas embolism.

Q. Describe the effects of pneumoperitoneum on other organ systems.

Ans. Renal effects: There is a decrease in renal blood flow and glomerular filtration rate due to the reduction in cardiac output. Renal vascular resistance may also increase due to the raised IAP. This can manifest as a reduction in intraoperative urine output. Patients with preexisting renal dysfunction are at risk of further deterioration. However, normally these changes are reversible and the urine output improves once IAP comes down at the end of surgery.

Splanchnic and hepatic blood flow: The effect of pneumoperitoneum on splanchnic and hepatic blood flow is unclear. Animal studies suggest that while hypercapnia has a vasodilatory effect, this is opposed by the vasoconstricting effect of the raised IAP.

Gastrointestinal effects: Raised IAP can predispose the patient to regurgitation and aspiration. However, it is believed that the changes in the IAP are transmitted to the lower esophageal sphincter, preventing this complication.

Neurologic effects: The combination of factors such as hypercapnia, head-low position, and elevated systemic vascular resistance can lead to an increase in intracranial pressure with a resultant decrease in cerebral perfusion pressure. This may be exaggerated in patients with ventriculoperitoneal shunts which do not have a unidirectional valve.

Ocular effects: A slight increase in intraocular pressure has been described, though its clinical significance is not clear.

Neuroendocrine effects: Increased levels of stress hormones. Increase in antidiuretic hormone (ADH) and aldosterone levels.

Q. What are the alternatives to pneumoperitoneum for laparoscopy?

Ans. The gasless laparoscopic technique avoids using any gas for insufflation, relying instead on an abdominal wall lift to create an intra-abdominal space at atmospheric pressure. It can be considered for elderly patients or those with cardiopulmonary problems. Hand-assisted laparoscopy is another form of laparoscopy.

Q. Describe the preoperative management of this patient.

Ans. The patient must have a thorough preoperative assessment. Investigations such as hemogram, liver and renal function tests, chest X-ray (CXR), and electrocardiogram (ECG) should be performed. If history or ECG is suggestive of cardiac disease, further testing in the form of two-dimensional (2D) echocardiography or

stress testing is indicated. If the patient has a history of gastroesophageal reflux, she should be started on proton-pump inhibitors in the preoperative period. Deep venous thrombosis prophylaxis with heparin or low-molecular weight heparins should be considered. Patients with prolonged jaundice should be evaluated for coagulopathy and for hepatic dysfunction secondary to long-standing obstruction.

Q. What monitors will you use for this patient?

Ans. Electrocardiogram, noninvasive arterial pressure monitor, airway pressure monitor, pulse oximeter, ETCO₂ concentration monitor, peripheral nerve stimulator (optional), and core body temperature monitoring should be used.

Q. In high cardiopulmonary risk patients, what additional monitors would you like?

Ans. For patients with compromised cardiopulmonary function, continuous arterial pressure, cardiac filling pressures, and blood gases monitoring is indicated. In these patients, the PaCO₂-ETCO₂ gradient is increased after CO₂ pneumoperitoneum, and ETCO₂ may not accurately reflect the PaCO₂ status.

Q. What is the anesthetic plan for this patient?

Ans. Balanced general anesthesia technique with endotracheal intubation and controlled ventilation is recommended. Nitrous oxide should be avoided. At induction, care should be taken during mask ventilation to avoid gastric insufflation, which increases the risk of trocar damage and may impede the surgical view. In cases of inadvertent gastric insufflation, the stomach must be aspirated before trocar placement to avoid gastric perforation. During pneumoperitoneum, controlled ventilation must be adjusted to maintain ETCO₂ at approximately 35 mm Hg.

Q. Can this patient be managed without intubation?

Ans. The use of supraglottic airways for laparoscopy is controversial. The first-generation devices do not fully protect against aspiration of stomach contents and are ordinarily used with lower peak inspiratory pressures. Second-generation devices have an additional gastric access and protect against aspiration of gastric contents. These devices have higher sealing pressures and may be used for laparoscopic surgery.

Q. Is it advisable to use nitrous oxide (N₂O) during this surgery?

Ans. It is recommended to avoid N₂O for two reasons. First, it diffuses into the abdominal cavity in concentrations sufficient to support combustion of intestinal gas; second, it will diffuse into CO₂/air bubbles, increasing their size and

the potential for an obstruction of pulmonary circulation. There is some concern about N_2O diffusing into the bowel and causing distension during laparoscopic surgery, but studies have shown no difference in operating conditions irrespective of whether N_2O was used or not.

Q. Can this procedure be done under regional anesthesia?

Ans. Regional anesthesia offers several advantages—quicker recovery decreased postoperative nausea and vomiting (PONV), less postoperative pain, shorter postoperative stay, cost-effectiveness, and fewer hemodynamic changes. Complications of general anesthesia such as sore throat, muscle pain, and airway trauma can be avoided. However, regional anesthesia will be suitable only if the patient is relaxed and cooperative and the surgical technique is gentle. The combined effect of pneumoperitoneum and sedation can lead to hypoventilation and arterial oxygen desaturation. Extensive sensory block is needed (T5 to L2) and shoulder tip pain due to diaphragmatic irritation may not be alleviated. This can be treated by intravenous fentanyl and by local spraying of the right diaphragmatic crus.

Q. What position will be used for this patient? Describe the effects of the operative positions used in laparoscopic surgery.

Ans. Laparoscopic cholecystectomy is usually carried out in the reverse Trendelenburg position, with the patient's arms by the side. This position helps by displacing the abdominal contents away from the surgical site, to facilitate surgical exposure.

For laparoscopic procedures depending on site of surgery either Trendelenburg (for pelvic surgeries) or reverse Trendelenburg (for upper abdominal surgeries) position is used. The effects of surgical positioning are:

- *Reverse Trendelenburg (head up):* Preload is decreased, resulting in lowered pressure [mean arterial pressure (MAP)]. Blood pooling in the lower extremities may increase the risk of venous thrombosis and pulmonary emboli. However, pulmonary function is improved.
- *Trendelenburg position (head down):* Cardiac output and central venous pressure increase. Pulmonary effects include impaired diaphragmatic function secondary to the cephalad displacement of abdominal viscera, resulting in decreased FRC, decreased total lung capacity, and decreased pulmonary compliance, predisposing the patient to developing atelectasis. Cephalad movement of the trachea may result in endobronchial intubation.

Q. What other problems can occur during positioning?

Ans.

- Nerve injuries can occur during positioning—the ulnar nerve when the upper limbs are adducted against the

body, the brachial plexus when using shoulder braces, and the common peroneal nerve in the lithotomy position.

- In steep head-up position, the patient must be securely strapped to prevent the patient from slipping off the table.
- Access to intravenous lines may be a problem and the use of extension tubing may be needed.

Q. During the surgery, the patient has a sudden hemodynamic collapse. What is the differential diagnosis and how will you manage the patient?

Ans. There are several causes of hemodynamic collapse during laparoscopy:

- *Hemorrhage:* Major vascular injuries can occur due to accidental insertion of the Veress needle or trocar into major vessels such as the aorta or common iliac vessels. Minor vascular injuries can involve the abdominal wall vessels.
- *Pneumothorax:* Pneumothorax can occur with the gas traversing into the thorax either through a tear in the peritoneum or parietal pleura during dissection, or due to rupture of preexisting emphysematous bulla. There can be associated pneumomediastinum or subcutaneous emphysema. An initially asymptomatic pneumothorax can progress to a tension pneumothorax with high peak airway pressures, decreased saturation, hypotension, and cardiac arrest.
- *Gas embolism:* Gas insufflated under pressure into a tear in a vessel can lead to gas embolism. This leads to hypotension, arrhythmias, and desaturation, and can progress to cardiac arrest. A sudden fall in $ETCO_2$ should raise suspicion of gas embolism. The treatment consists of deflating the abdomen, ventilation with 100% O_2 for carbon dioxide elimination, central venous catheter (CVC) placement for aspiration of gas (inserting CVC after air embolism is generally of no use as air has already entered pulmonary circulation and cannot be aspirated from CVC), and cardiopulmonary resuscitation.

Q. How will you manage this patient's postoperative pain?

Ans. Pain following laparoscopic cholecystectomy can arise from the incision site (incisional pain), visceral structures (abdominal pain) or referred from the subdiaphragmatic region as shoulder pain.

Shoulder pain is often mild in intensity and is due to irritation of the diaphragm by residual gas. It can be minimized by completely evacuating all gas from the abdominal cavity prior to closure.

Abdominal pain can occur due to stretching of the parietal peritoneum, release of inflammatory mediators of

pain, and irritation produced by the blood. Recommended modalities of prevention and treatment include the use of suction to remove any blood and insufflated gas at the end of surgery as well as by instillation of normal saline to "dilute" any local pain mediators.

Local infiltration of local anesthetic into the incision and trocar sites provides good pain relief. The role of intraperitoneally injected local anesthetic to provide pain relief has been debated. It should be remembered that toxicity can occur when larger doses are used.

Blocks like the transversus abdominis plane block may also be used for postoperative analgesia.

The most effective pain relief can be obtained by combining opioids, local anesthetics, and nonsteroidal anti-inflammatory drugs (NSAIDs) into balanced analgesia.

Q. If this patient had heart disease, would laparoscopy be safe?

Ans. There are various factors which stress the cardiovascular system during laparoscopy. The reduction in preload causes the heart rate to increase to maintain cardiac output. There is increased afterload, as a result of the elevated systemic vascular resistance. The ventricular wall tension increases, reducing coronary blood flow thus leading to myocardial ischemia. Sympathetic stimulation may occur due to hypercapnia. Therefore, if the patient is intolerant of tachycardia, and cannot compensate for drop in venous return, laparoscopy may be dangerous.

Q. What measures need to be taken during the anesthetic management of cardiac patients undergoing laparoscopy?

Ans. Patients with a profile suggesting depleted intravascular volume experience the most severe hemodynamic changes. Preload augmentation before peritoneal insufflation partially offsets hemodynamic deterioration:

- Invasive hemodynamic monitoring is essential.
- It must be remembered that central venous pressures may not be reliable in the presence of a pneumoperitoneum.
- Preload optimization is necessary to prevent hypotension.
- Gas insufflation must be slow, and IAP must be kept as low as possible.
- Vasodilators can be used to correct or prevent the increase in afterload; however, it should be kept in mind that this can lead to further drop in venous return and cardiac output.
- An inotropic agent such as dobutamine can be added to improve ventricular function.
- A diuretic may be needed at the end of the surgery.
- Gasless laparoscopy may be a useful alternative in patients with ischemic heart disease who cannot tolerate CO₂ pneumoperitoneum.

Q. What are the specific points in managing anesthesia for a patient with chronic obstructive pulmonary disease, who is posted for laparoscopic cholecystectomy?

Ans. Minimally invasive surgery offers advantages over open surgery in terms of less tissue handling, and less postoperative pain, leading to better respiratory function in the postoperative period. Preoperative respiratory optimization, with bronchodilator therapy in responsive patients, treatment of super-added infection, and chest physiotherapy is indicated. During ventilation of these patients, adequate expiratory time should be maintained to prevent air-trapping and development of auto-PEEP. Airway pressures may be high; it may be necessary to limit hyperventilation and accept modest levels of hypercapnia. ET-CO₂ may not be an accurate estimate of Pa-CO₂ because the Pa-CO₂ to ET-CO₂ gradient increases.

Q. Is laparoscopy safe during pregnancy?

Ans. Laparoscopy can be performed during pregnancy, especially during the second trimester because organogenesis is complete, the uterine size is not large enough to interfere with the operative field, and the risk of spontaneous abortion is low.

Q. What advantages does laparoscopy offer for the parturient?

Ans.

- Less exposure of fetus to potentially toxic agents.
- Smaller incisions with less pain, and therefore reduced requirement for postoperative analgesics.
- More rapid recovery and mobilization.

Q. What are the physiological changes in pregnant patients after pneumoperitoneum?

Ans. Pregnancy is associated with physiological changes in the cardiovascular and respiratory system. These include a cephalad shift of the diaphragm leading to decreased FRC and compression of the inferior vena cava by the gravid uterus leading to hypotension. Laparoscopy can further exacerbate these changes, making the parturient more susceptible to hypoxemia and hypotension. Head-low position will worsen the ventilation, though it improves venous return. Head-high position can improve ventilation, but it will further decrease venous return and cardiac output. Hyperventilation to treat hypercarbia should be controlled as maternal respiratory alkalosis will lead to decreased uteroplacental perfusion.

Q. What specific measures will you follow while anesthetizing a pregnant patient undergoing laparoscopy?

Ans.

- Preoperative discussion with the obstetrician
- Pneumatic compression stockings, as these patients are more prone for deep vein thrombosis (DVT)

- Rapid-sequence induction with aspiration prophylaxis
- Position with uterine displacement
- Fetal heart rate monitoring if >16 weeks gestational age, in addition to other monitors
- Orogastric tube to deflate the stomach and improve visualization
- Limit IAP to 12–15 mm Hg
- Routine tocolysis is not recommended—isoxsuprine or terbutaline may be used in specific cases.

Q. What are the contraindications for laparoscopy?

Ans. Absolute contraindications for laparoscopy include:

- Shock
- Significantly raised ICP
- Retinal detachment
- Right-to-left shunts.

Though laparoscopy is not absolutely contraindicated, special care must be taken in patients with bullous emphysema or patients having ventriculoperitoneal shunt, hypovolemia, congestive heart failure or other severe cardiopulmonary disease, and coagulopathy.

Q. Can laparoscopic surgery be performed in children?

Ans. Laparoscopy has been used safely for procedures such as fundoplication, hernia repair, and orchidopexy in all age groups of children, including neonates. However, to minimize the cardiorespiratory effects of raised IAP, insufflation pressures should be limited to 5–10 mm Hg in neonates and infants and 10–12 mm Hg in older children.

Q. What is robotic surgery device?

Ans. A robotic device is a powered, computer controlled device with artificial sensing system that can be programmed for positioning and manipulations of surgical instruments.

Q. What are the advantages of robotic surgery over laparoscopic surgery?

Ans. Robotic surgery offers the benefits of laparoscopic surgery, like improved cosmesis, less postoperative pain, and faster recovery with shorter hospital stay and in addition robotic surgery also overcomes some of the shortcomings of conventional laparoscopic surgeries such as the assistant-dependent unstable video camera platform, 2D view, restricted ergonomics of the surgeon, and instruments with limited degrees of freedom, and the absence of wrist gear. Robotic systems present 3D views with magnification and tools with 7° of freedom that are capable of duplicating hand movements with high accuracy.

Q. What are the anesthetic considerations of robotic surgeries?

Ans.

- Restricted availability of space in the operating room (OR) due to bulky equipment.

- Once the robot is docked, access to the patient is limited; therefore, anesthesia circuits and tubings must be securely fastened to prevent disconnections; intravenous lines should have extension tubings to allow access; additional monitoring (e.g., arterial blood pressure monitoring) must be planned in advance.
- Operating room personnel must be trained to respond in an emergency—since the procedure of undocking the robot and moving it away from the patient takes time.
- Any type of patient movement during the surgery is not allowed. Movement of the patient while robotic instruments are docked can lead to tearing or puncturing of internal organs and vasculature, with serious consequences.
- Positioning for surgery is more extreme than for conventional laparoscopic surgery. In the steep reverse Trendelenburg position, the patient is more likely to slip and restraints need to be used. These extreme positions also cause physiological changes.
- Bulky robotic arms, extreme positioning, and prolonged surgery durations place patients at risk of positioning injuries.

Q. What are the specific anesthetic considerations of various types of robotic surgeries?

Ans.

Robotic-assisted Urological Surgeries

Robot-assisted radical prostatectomy (RARP) is one of the most commonly performed robotic surgery. The usual blood loss is 150–250 mL. Robot-assisted partial nephrectomy (RAPN) is associated with a significant reduction in blood loss, surgical complications and less risk of renal damage as it shortens the “warm ischemic time.” The anesthetic concerns associated with robotic prostatectomy are mainly due to steep Trendelenburg position and CO₂ pneumoperitoneum. RARP requires a much steeper Trendelenburg position and a greater pressure of CO₂ pneumoperitoneum. The retroperitoneal dissection increases the absorption of CO₂.

The combination of steep Trendelenburg position and CO₂ pneumoperitoneum affects the patient synergistically. Patients are prone to develop ventilation-perfusion mismatch, atelectasis, and pulmonary edema. Ventilating with a tidal volume of 6–8 mL/kg, a PEEP of 4–7 cmH₂O and I:E ratios of 2:1 or 1:1 provide better oxygenation and lower PaCO₂ levels and help to prevent atelectasis. Pressure-controlled ventilation produces better compliance and lower peak inspiratory airway pressure, compared with volume-controlled ventilation. Many authors recommend placing thoracic epidural as it is found to lower peak inspiratory pressure, enhance dynamic compliance, allow better oxygenation and lower concentrations of lactate through more profound muscle block.

These combined effects of steep Trendelenburg position and CO₂ pneumoperitoneum lead to an increase in myocardial oxygen demand, and therefore one must be careful in patients with a reduced cardiac reservoir or impaired baroreflex.

The steep Trendelenburg position raises the intracranial pressure and intraocular pressure (IOP) with surgical duration and ETCO₂ being the main predictors. IOP increases by an average of 13 mm Hg after being positioned in the 25° Trendelenburg position with 15 mm Hg of CO₂ pneumoperitoneum, compared with the preinduction values.

The period of transection of the deep dorsal venous plexus, rather than the initial insufflation, is more likely to be the risky period for gas embolism.

Swelling of the face and upper airway with venous stasis in the head and neck is common due to prolonged steep Trendelenburg position. Mildly restrictive fluid management is advocated to reduce facial edema and reduce the urine output that obscures the surgical field. Before extubation, the significance of upper airway edema should be evaluated properly. Lingual, buccal nerve neuropathies, ischemic optic neuropathy and corneal abrasions are common and necessary precautions should be taken. RARP usually requires the patient in a lithotomy and steep Trendelenburg position. Proper padding of bony prominences should be done to prevent peripheral nerve injuries.

Robotic-assisted Gynecological Surgeries

Hysterectomy, myomectomy, tubal re-anastomosis, radical hysterectomy, lymph node dissection, and sacrocolpopexy are commonly performed using robotic surgical systems. Most of the anesthesia concerns are the same as for RARP because of the similar positioning and pneumoperitoneum. In gynecological robotic surgeries less-steep Trendelenburg position, compared with urological surgery, is permitted.

Transoral Robotic Surgeries

Radical tonsillectomy, tongue base resection, supraglottic laryngectomy, and phonomicrosurgery can be performed using the robotic surgical system. Exclusion criteria being pediatric diseases, lesions invading the mandible, and dental procedures. Advantages of transoral robotic surgery (TORS) are better recovery of postoperative quality of life, such as speaking and eating. Anesthesia considerations for TORS are quite similar to tonsillectomies and laser excision of laryngeal lesions. However, few things need to be considered. Patient is turned 180° away from the anesthesia workstation making induction more complicated, and a large robot is placed in the vicinity of the patient's head.

Also many times the patient is placed in suspension laryngoscopy. Eyes should be secured with safety goggles and teeth should be protected with a dental guard in order to avoid injury during insertion of the robotic arm into the intraoral space. Surgeon may suture the endotracheal tube to the patient's face, thus a wire-reinforced tube is commonly used. Invasive lines are usually not needed. However, the anesthesiologist should be aware of the risk of lingual artery rupture, and the need of dissection of the carotid artery plane in some surgeries. In prolonged and difficult surgeries, a tracheostomy or postoperative intubation may be needed.

Robotic Thoracic Surgeries

Thymectomy, mediastinal mass excision, fundoplication, esophagectomy, and pulmonary lobectomy are commonly performed robotically. The key issue for anesthesia during one-lung ventilation and capnothorax is maintaining stable hemodynamics and oxygenation. It is important to select patients carefully for thoracic robotic surgeries. For thymectomy, the patient is placed in an incomplete side-up position, at a 30° right or left lateral decubitus position. The arm of the elevated side is positioned at the patient's side, as far back as possible, so the surgeon can have enough space for the robotic arms. This increases the chances of brachial plexus injury.

CO₂ insufflation pressure of 10–15 mm Hg is applied to keep the lung away from the surgical field, but can obstruct venous return and cause profound hypotension.

Because lung isolation is mandatory for esophagectomy, thymectomy, or mediastinal tumor resection, left-sided double lumen tubes is usually used. The key issue during one-lung ventilation and capnothorax is maintaining stable hemodynamics and oxygenation.

Long monitoring lines and anesthesia circuits, extensions for intravenous lines are necessary, and injection ports should be in accessible locations.

During the surgery, compression of cardiac or major vascular structures may result in hemodynamic instability, with ECG changes. Hence, invasive monitoring is necessary.

There is a high risk of positioning neuropathy during robotic thoracic surgery. Inadequate padding or positioning and inadvertent robotic arm placement can cause external nerve compression.

Robotic Surgery in Pediatric Patients

Robot-assisted surgeries have been increasingly performed in pediatric patients. Some examples being pyeloplasty, patent ductus arteriosus (PDA) closure, nephrectomy, Bochdalek hernia repair, Kasai portoenterostomy, and choledochal cyst excision.

In the preoperative anesthesia workup, it is important to rule out congenital malformations as many of the congenital abnormalities of the genitourinary tract, often treated with MIS, may be associated with abnormalities in central nervous system, respiratory tract and airways.

In children, the abdominal wall is thinner than in adults, inadvertent visceral injury during port introduction and instrument manipulation should always be kept in mind. Lower insufflation pressures of 4–12 mm Hg is usually sufficient to visualize intraperitoneal structures and create operating space as far as the prepubertal abdominal wall is considered. The associated increase of IAP, along with absorption of CO₂ and surgical positioning requirement, may result in neuroendocrine and mechanical impact capable of depress cardiopulmonary function.

■ SUGGESTED READING

1. Baroncini S, Gentili A, Pigna A, Fae M, Tonini C, Tognù A. Anesthesia for laparoscopic surgery in paediatrics. *Minerva Anesthesiol.* 2002;68:406-13.
2. Gerges FJ, Kanazi GE, Jabbour-Khoury SI. Anesthesia for laparoscopy: a review. *J Clin Anesth.* 2006;18:67-78.
3. Hayden P, Cowman S. Anaesthesia for laparoscopic surgery. *Contin Educ Anesth Criti Care Pain.* 2011;11:177-80.
4. Joris JL. Anesthesia for laparoscopic surgery. In: Miller RD. *Miller's Anesthesia*, 7th edition. Philadelphia: Churchill Livingstone; 2010.
5. Kakar PN, Das J, Roy PM, Pant V. Robotic invasion of operation theatre and associated anaesthetic issues: A review. *Indian J Anaesth.* 2011;55:18-25.
6. Lee JR. Anesthetic considerations for robotic surgery. *Korean J Anesthesiol.* 2014;66:3-11.
7. Perrin M, Fletcher A. Laparoscopic abdominal surgery. *Contin Educ Anesth Criti Care Pain.* 2004;4:107-10.

Colles' Fracture

Parmanand Jain, Aparna Chatterjee

A 60-year-old diabetic patient fell in the bathroom on an outstretched hand and sustained a wrist fracture.

Q. What are the main features of Colles' fracture?

Ans. Fracture of the distal radius as described by Abraham Colles in 1814 is known as Colles' fracture. There are several types of distal radius fractures such as Colles' fracture, Smith's fracture, Barton's fracture or Chauffeur's fracture (so called because the crank used to start old cars often kicked back and broke the Chauffeurs' wrists with a particular pattern). Most of these names are applied to specific patterns of distal radius fracture but confusion exists. "Colles' fracture" is used as a generic term for distal radius fracture.

The patient usually presents with a history of fall on an outstretched hand and has a typical deformity of the wrist known as dinner-fork deformity.

Q. What is the surgical management of Colles' fracture?

Ans.

Management of Fractures

Conservative: The principle of conservative management is closed reduction of the fracture by manipulation and maintenance of reduction with the application of a cast:

- *Closed reduction of the fracture:* Closed reduction means the restoration of the normal anatomical alignment of fragments by application of an external force in a direction opposite to that which produced the injury without making incision on the skin. Adequate anesthesia is of utmost importance for closed reduction of a Colles' fracture.
- *Maintenance of reduction:* In the majority of fractures, the maintenance of the alignment of the fragments is done by immobilization with plaster of Paris (POP) cast.
- Manipulation generally includes first placing the arm under traction and unlocking the fragments. The deformity is then reduced with appropriate closed manipulations (depending on the type of deformity)

reduction, after which a splint or cast is placed and an X-ray is taken to ensure that the reduction was successful. The cast is usually maintained for about 6 weeks.

Open (Surgical) Reduction and Internal Fixation

Majority of fractures can be satisfactorily managed by manipulation and closed reduction. However, in case of some fractures there is an inherent instability of the fragments or a tendency for delayed union or nonunion. Such fractures are treated by open reduction of the fracture by surgical method and internal fixation.

Q. What is the anesthetic management of Colles' fracture?

Ans. Closed reduction of a distal radius fracture can be performed under general anesthesia, intravenous regional anesthesia (Bier's block/IVRA); intravenous (IV) sedation, peripheral nerve block local and hematoma block (HB).

The anesthesia options for patients undergoing an open surgery for reduction of a Colles' fracture would include regional (peripheral nerve block) anesthesia or general anesthesia.

The type of anesthetic technique used depends on the nature and duration of the surgery, patient's health, any medical conditions, and preferences of the patient, surgeon, and anesthetist.

Q. What are the various regional anesthesia or nerve block techniques employed for Colles' fracture?

Ans. The nerve blocks that can be employed in increasing order of preference are: IVRA, brachial plexus block (infraclavicular or axillary), and nerve blocks at the elbow.

Intravenous regional anesthesia block (Biers' block): Described by August Bier in 1808, it involves injection of local anesthetic (LA) (lignocaine) through an IV cannula sited on the affected hand. Two tourniquets are placed

around the upper arm. The arm must be exsanguinated with an Esmarch bandage before injection of the LA. After exsanguination of the hand, forearm, and upper arm to the distal tourniquet, the distal and proximal tourniquets are inflated. The distal tourniquet is deflated initially, and the first portion of the procedure is performed under tourniquet control with the proximal tourniquet.

The tourniquets should be inflated to at least 100 mm Hg above systolic blood pressure.

Onset of analgesia: 5 minutes, complete analgesia within 10 minutes.

During the procedure, patients often become less tolerant of the proximal tourniquet at 40 minutes. At this point, the distal tourniquet is inflated over the anesthetized area. The painful proximal tourniquet can then be deflated. At the conclusion of the procedure, the tourniquet is deflated and patient is closely observed for at least 10 minutes.

In case the procedure is of a shorter duration, the tourniquet should remain inflated for a minimum of 25 minutes after LA injection even if surgery has been completed to minimize the possibility of a systemic toxic reaction. If block is to be reinstituted within 30 minutes after tourniquet release, 50% of the initial dose of the LA can be used.

However, one has to remember that it may be difficult to insert an IV cannula since there could be a lot of swelling on the hand. Pain due to the fracture may make IV insertion and exsanguination of the arm difficult.

Complications

- Leaking or accidentally deflated tourniquet
- Large volume of LA introduced into circulation, with symptoms and signs of toxicity.

Drugs used for Intravenous Regional Anesthesia

- *Nonadrenaline containing solutions of:*
 - *Prilocaine 0.5%:*
 - Drug of choice. Least toxic and largest therapeutic index
 - 40 mL for upper limb, 60 mL for lower limb
 - *Maximum dose for a 70 kg adult:* 400 mg (approximately 6 mg/kg)
 - *Lignocaine 0.5%:* Alternative—
 - *Maximum dose for a 70 kg adult:* 250 mg (3 mg/kg)
 - *Bupivacaine is contraindicated.*

Advantages:

- Simple, easy technique
- Rapid onset of action
- 99–100% success rate
- Low cost
- Can be used for outpatient surgery.

Disadvantages:

- No postoperative analgesia
- Limited to procedures lasting <1 hour
- Cannot be used where tourniquet is contraindicated, e.g., sickle cell disease
- Local anesthesia toxicity in case of tourniquet failure.

Contraindications:

- Patient refusal
- Raynaud's or peripheral vascular disease
- Sickle cell disease
- Crush injuries
- *Children (age <10 years):* Intraosseous vessels in prepubertal patients may allow the LA to bypass the tourniquet
- Uncontrolled hypertension
- Obese (tourniquet may not compress the arteries effectively)
- Surgery lasting more than 2 hours.

Brachial Plexus Block

The brachial plexus provides motor and sensory innervations of the upper limb. It is formed by the anterior primary rami of C5–8, together with T1. There is also some contribution from C4 and T2 at times.

It can be blocked by different approaches, namely:

- *Interscalene:* For surgery on the shoulder, upper arm and elbow
- *Supraclavicular:* Surgery on the elbow, forearm and hand
- *Infraclavicular:* Surgery on the forearm, wrist and hand
- *Axillary:* Hand, wrist and lower forearm surgery.

For surgery on the distal radius-ulna (lower forearm), such as that with a Colles' fracture; an infraclavicular or an axillary brachial plexus block would suffice.

Infraclavicular Brachial Plexus Block

Block occurs at the level of the cords and offers the theoretical advantages of a minimal risk of pneumothorax while blocking the musculocutaneous and axillary nerves.

Three techniques have been described:

1. The classical (Raj) approach
2. The coracoid approach
3. The vertical infraclavicular approach.

Classical approach: The needle is inserted 2 cm below the midpoint of the inferior clavicular border and advanced laterally, directed toward the axilla, away from the lung and requires three-dimensional orientation of the pyramid-shaped anatomy of the axilla to be successful. A nerve stimulator can be used to identify the plexus. Marking a line between the C6 tubercle and the axillary artery with the arm abducted is helpful in tracing the course of the plexus. Incremental injection of 20–30 mL of solution is

sufficient after the needle is correctly placed. The success rate is improved with a distal motor response. It is associated with a minimal risk of pneumothorax and good reliability.

Coracoid approach: The needle is inserted 2 cm medial and 2 cm caudal to the coracoid process, directed posteriorly and perpendicular to the skin. However, the insertion site is quite lateral and may result in the absence of blockade of the musculocutaneous nerve, thus removing the major advantage of this approach over the simpler axillary block.

Vertical infraclavicular approach: Recently, the vertical infraclavicular block has been described. The needle entry point is immediately below the clavicle at a point midway between the sternal notch and the ventral apophysis of the acromion. The needle must be advanced in a vertical direction to a maximum depth of 4 cm.

Axillary Brachial Plexus Block

The axillary approach is the most popular because of its ease, reliability, and safety. Blockade of the musculocutaneous nerve is not always produced with this approach but it can be supplemented at the level of the axilla or at the elbow. The injection is made at the level of the terminal branches; hence the missed segment(s) show a nerve territory distribution and not a dermatomal pattern, as is the case when the injection is placed at the level of the roots.

The patient is made to lie supine with the arm to be blocked placed at right angle to the body with the elbow flexed to 90°. The needle is introduced just superior to the pulsation of the axillary artery at the lateral border of pectoralis major muscle. Digital pressure is often applied just distal to the injection site to aid proximal spread of the LA. A typical volume of LA required is 30–40 mL. The use of a large volume (40 mL) of LA ensure blockade of the musculocutaneous nerve for the tourniquet area.

Transarterial techniques have been described; the main complication from this technique is a hematoma formation.

Researchers have compared multiple and single injection techniques. A multiple injection technique involves identifying two, three or four separate terminal nerves in the plexus and injecting a small amount of LA around each of them. It increases the success rate in blocking the musculocutaneous nerve but there is a slightly higher chance of neuropraxia after the block.

Other Brachial Plexus Techniques

Interscalene Brachial Plexus Block

Ideal block for surgery on the shoulder. Block occurs at the level of the upper and middle trunks. Blockade of the inferior trunk is usually incomplete and the ulnar nerve may be missed and may have to be supplemented if used for forearm and hand surgery.

With the patient in supine position, arm by their side and the head turned away from the side to be blocked, the posterior border of the sternocleidomastoid muscle is identified by making the patient briefly lift his/her head. The interscalene groove can be palpated by rolling the fingers posterolaterally from this border over the belly of the anterior scalene muscle into the groove. One can also ask the patient to sniff forcefully, this tenses the scalene muscles and the interscalene groove becomes prominent. A line extended laterally from the cricoid cartilage and intersecting the interscalene groove indicates the level of the transverse process of C6.

The point of insertion of the needle is the interscalene groove, lateral to the sternomastoid at the level of the cricoids cartilage. The needle is directed medially, caudally and posteriorly, i.e., toward the transverse process of the C6 vertebra. If the needle is directed cranially, there is a risk of accidental epidural, intrathecal or intravascular injection. 30–40 mL of LA solution is injected.

Complications:

- *Phrenic nerve block:* Avoid in patients with contralateral phrenic nerve palsy
- Recurrent laryngeal nerve block
- Horner's syndrome
- Accidental epidural or intrathecal injection
- Accidental injection into the vertebral artery.

Supraclavicular Brachial Plexus Block

Blockade at the distal trunk–proximal division level. At this point, the plexus is compact and a small volume of solution produces rapid onset of reliable blockade of the brachial plexus.

Patient is placed supine, head turned away from the side to be blocked. The needle is inserted immediately posterior and lateral to the subclavian artery pulsations behind the mid-clavicle. The needle is then directed caudally, medially and posteriorly to the upper surface of the 1st rib and walked along the rib. About 8–10 mL of LA is injected per division. A total of 20–30 mL of solution is required. A reliable supraclavicular blockade requires elicitation of a paresthesia or motor response. The median nerve may be missed in this block.

The supraclavicular approach is best avoided if the patient is uncooperative or cannot tolerate any respiratory compromise. Other complications include frequent phrenic nerve block (40–60%), Horner's syndrome, and neuropathy.

Bilateral blocks should be avoided.

Block of Nerves at the Elbow (Antecubital Fossa)

Elbow blocks are rarely used alone for forearm procedures. Usually used to supplement patchy brachial plexus blocks. Knowledge of the territory innervated by each nerve is

essential so that the appropriate nerves can be blocked for the operation.

The following six nerves can be blocked in this area:

1. Ulnar nerve
2. Median nerve
3. Radial nerve
4. Medial cutaneous nerve of forearm
5. Lateral cutaneous nerve of forearm
6. Posterior cutaneous nerve of forearm.

Approximately 5–10 mL of LA is appropriate for blocking each of these nerves.

These blocks are easy to learn and perform but are of shorter duration.

Ulnar nerve: The ulnar nerve is easily accessible at its subcutaneous position posterior to the medial epicondyle; blockade at this site is associated with a high incidence of neuritis. The nerve is surrounded by fibrous tissue at this point; there is a high risk of intraneural injection hence the nerve is blocked proximal to and not in the ulnar groove. Use of a very fine needle along with a small volume of solution (1 mL) diminishes the risk. The nerve can be satisfactorily blocked with 5–10 mL of solution at a site 3–5 cm proximal to the elbow. The LA should be injected in a fan-like fashion without elicitation of a paresthesia.

Median nerve: Lies medial to the brachial artery, where it emerges medial to the biceps tendon. It can be blocked at a point 1–2 cm proximal to the flexor skin crease at the antecubital fossa. Approximately 3–5 mL of LA solution is injected after eliciting a paresthesia. If no paresthesia is obtained, the solution can be injected in a fanlike pattern medial to the palpated artery.

Radial nerve: The radial nerve can be blocked at the elbow as it passes over the anterior aspect of the lateral epicondyle. The intercondylar line and lateral edge of the biceps tendon are marked. A 22-gauge, 3–4 cm needle is inserted at a point 2 cm lateral to the biceps tendon and advanced until bone is encountered. 3–5 mL of solution is injected in a fanlike fashion.

Medial cutaneous nerve of forearm: Innervates the skin on the medial side of the forearm and can be blocked at the elbow after a median nerve block by withdrawing the needle and directing it proximally injecting the LA subcutaneously between the head of the pronator teres muscle and the medial border of the biceps tendon.

Lateral cutaneous nerve of forearm: The musculocutaneous nerve terminates as the lateral cutaneous nerve of the forearm and innervates the skin of the lateral side of the forearm. It can be blocked 1 cm proximal to the

intercondylar line immediately lateral to the biceps tendon. Fanlike infiltration of 3–5 mL of solution subcutaneously at this site provides excellent anesthesia of this nerve.

Posterior cutaneous nerve of forearm: It is the proximal branch of the radial nerve and innervates the skin on the posterior aspect of the forearm. It can be blocked by injecting the LA solution subcutaneously from the lateral epicondyle of the humerus to the olecranon.

Ultrasound Guidance for Peripheral Nerve Blocks

Peripheral nerve blocks are routinely performed without visual guidance. The needle insertion is guided only by surface anatomical landmarks. These techniques have a failure rate of approximately 20% because of incorrect needle or LA placement. Blind techniques are also associated with an increased risk of nerve injuries due to direct impalement or intraneural injection of the drug.

Use of a nerve locator increases the likelihood of placing the needle in close proximity to the nerve, however, does not completely eliminate the risk of nerve impalement and postoperative neuritis. Multiple attempts at needle placement can lead to a lot pain and discomfort for the patient.

Ultrasound is being increasingly used over the last several years to assist nerve blocks. Ultrasound allows direct visualization of peripheral nerves, the block needle, and LA distribution and has proven to be very useful to guide targeted drug injections and catheter placement (95–99% success rate).

Advantages:

- Nerves are clearly visualized on ultrasound.
- Shows the exact location of the nerves, more so valuable in patients with anatomical variations.
- Provides a real-time imaging, guiding needle placement. Allows continuous adjustments in direction and depth.
- Helps identify surrounding structures such as vessels and pleura, and avoid them while still positioning the needle close to the nerve.
- Local anesthetic spread can be visualized at the time of injection, allowing repositioning in case of an incomplete spread.
- Lesser volume of LA required for a nerve block, as it is more precise.
- Improves the onset time, success rate and quality of the block as compared with the nerve locator techniques.
- May reduce the number of attempts and potentially the risk of nerve injury, though it has not been proven.

Proficiency with the use of ultrasound for nerve blocks, however, requires training, practice, and familiarity with sonoanatomy.

Q. Approximately 15 minutes after giving Bier's block patient became hypotensive and restless. What is your diagnosis and how will you manage?

Ans. After Bier's block or regional block, one should have a very high index of suspicion for development of LA toxicity.

Signs of Local Anesthetic Toxicity

- Perioral paresthesia (lips, tongue, nose)
- Hypotension—may be caused by hypoxemia following central apnea, direct myocardial depression or vasodilatation
- Transient desaturation, cyanosis
- Arrhythmias—resistant ventricular arrhythmias (e.g., with bupivacaine)
- Dizziness, tremors
- Loss of consciousness, convulsion.

Management

Call for help: Reinflate cuff if it has been deflated—

- Airway—secure airway, 100% O₂, lateral position
- Breathing—ventilate
- Circulation—IV fluids, vasopressors, cardiopulmonary cerebral resuscitation (CPCR) if cardiac arrest
- Convulsions—IV diazepam, lorazepam, thiopentone.

SUGGESTED READING

1. Miller RD. Miller's Anesthesia, 7th edition. Philadelphia: Churchill Livingstone; 2009.
2. Wells M. Local and Regional Anesthesia in the Emergency Department Made Easy. Philadelphia: Churchill Livingstone; 2010.
3. Yentis SM, Hirsch NP, Smith GB. Anesthesia and Intensive Care A-Z, 4th edition. Philadelphia: Churchill Livingstone; 2009.

Kyphoscoliosis

Parmanand Jain, Sukhada Savarkar, Atul Prabhakar Kulkarni

A 16-year-old boy is posted for surgical correction of kyphoscoliosis.

Q. What is kyphoscoliosis?

Ans. The spine normally curves posteriorly in the thoracic (kyphosis) and anteriorly in the lumbar (lordosis) regions. Scoliosis refers to the lateral curvature of the spine. Curves are structural (lack normal flexibility and do not correct with bending toward the convexity or lying supine) or nonstructural (will resolve correctly with bending toward the convexity or in the supine position) due to limb length discrepancy. Scoliosis is sometimes associated with a kyphosis (hunchback) and lordosis (swayback).

Q. How do you classify scoliosis?

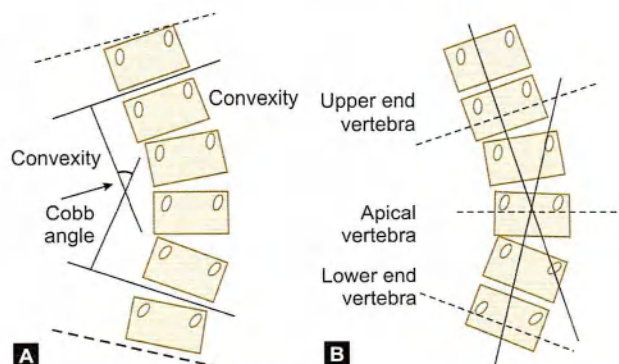
Ans. Scoliosis is classified as follows:

- **Idiopathic scoliosis:** The cause is obviously unknown, however, genetic inheritance, central nervous system dysfunction, and nutrition have all been blamed for causation. This type accounts for 70% cases. In this type, progression is much faster in girls; however, incidence is similar in both sexes. This is further divided into infantile (0–3 years), juvenile (4–10 years), and adolescent (10 years or more).
- **Congenital scoliosis:** It occurs due to an insult during the intrauterine period and is associated with other congenital anomalies (cardiac and urinary). This is further subclassified as open (meningomyelocele) and closed congenital scoliosis. Closed congenital scoliosis may occur due to: (1) failure of vertebral development that may be partial or complete and unilateral, and (2) unilateral or bilateral failure of segmentation of vertebrae.
- **Acquired scoliosis:** This may occur due to a variety of reasons. This includes:
 - Neuromuscular causes (muscular dystrophy and cerebral palsy)
 - Mesenchymal disorders such as rheumatoid disorders and osteogenesis imperfecta
 - Neurofibromatosis
 - Trauma
 - Vertebral tumors.

Q. How is the severity (degree) of scoliosis measured?

Ans. When the muscles responsible for vertebral alignment and weight transfer downward to pelvis weaken, a C-shaped scoliosis curve develops (primary curve). The balance system tries to compensate for the misalignment. A lean to the left at the top shifts the center of gravity to the left. To prevent tipping over, the lower spine is shifted to the right in an attempt to keep the center of gravity over the pelvis (compensatory secondary curve). The thoracic and lumbar regions are the most frequent sites of the primary curve.

In the Lippman–Cobb method (**Fig. 1A**), the first and last vertebrae that are most severely tilted toward the concavity are determined. Horizontal lines are drawn, one at the superior border of the upper end vertebra and another at the lower border of the lower end vertebra. A line perpendicular to these two lines is drawn and the intersecting angles are measured as shown in **Figure 1A**.



Figs. 1A and B: (A) Lippman–Cobb method; (B) Risser–Ferguson method.

This method is called the Lipmann–Cobb method and the angle as Cobb angle. Another method is the Risser–Ferguson method. In this method, the degree of scoliotic curvature is measured from the angle formed by the intersection of lines drawn from the middle of upper and lower end vertebrae to the center of apical vertebra (**Fig. 1B**). The Cobb angle is the most commonly used method in clinical practice.

Q. What are the aims and indications for surgery in a patient with kyphoscoliosis?

Ans. The aim of surgery is to prevent progression of the curvature of the spine, to maintain posture, and to prevent progression of the pulmonary dysfunction. The indication for surgical intervention is documented progression of the scoliotic curves at any age. For scoliosis that is not due to neuromuscular disease, an angle of 40° may be indication for surgery, but for most of those with neuromuscular problems like Duchenne, this curvature is reached after the respiratory muscles are found to be too weak for safe surgery. Hence, in such patients; a 25° curve is taken as a cutoff point instead.

Nowadays, many patients have both anterior and posterior procedures performed either under a single anesthetic or in patients with a more severe or rigid curve. The procedures are so major that they have to be staged as anterior followed by a subsequent posterior fusion. These sequential operations are performed within a specified time frame to optimize the results of stage 1 with those of stage 2. Anterior fusion involves thoracotomy or thoracoscopy. The thoracoscopic approach avoids cutting the chest/shoulder musculature, thus decreasing the morbidity of anterior spinal surgery.

Q. Discuss preoperative respiratory assessment and optimization in patients undergoing kyphoscoliosis correction surgery. What other systemic abnormalities are likely? Which patients are likely to need elective postoperative ventilation?

Ans. Preoperative considerations for patients undergoing major reconstructive surgery of the spine needing careful attention are given below.

These patients have extrinsic restrictive lung disease with a reduction in total lung and vital capacity. Spirometry will reveal the extent of restrictive disease and presence of obstructive element, if any. Reduction is worse with increasing spinal deformity. Respiratory mechanics is also worsened by the surgical correction of spinal deformity which alters the elastic forces acting on the chest wall. Surgery also leads to fluid shifts and third spacing, thus further compromising ventilatory capacity. It must be remembered that anesthesia and surgery further worsen the vital capacity. These patients also have increased pulmonary vascular resistance and ventilation and perfusion (V/Q) mismatch, which makes them more prone for hypoxia. All these factors must be carefully weighed to decide on the need for elective postoperative ventilation. Vital capacity <

40% almost always necessitates postoperative ventilatory support. Preoperative optimization involves teaching the patients breathing exercises, incentive spirometry, and breath holding techniques. Bronchodilator therapy (if obstructive element present) and hydration may be necessary to loosen secretions. The risk of postoperative respiratory failure due to pneumonia can be minimized by ensuring proper postoperative analgesia, which will allow good cough and removal of retained secretions.

Associated congenital heart disease and mitral valve regurgitation must also be ruled out. The presence of preoperative neurodeficit must be carefully documented to prevent postoperative medicolegal issues. The presence of musculoskeletal abnormalities makes them prone to abnormal response to muscle relaxants.

Q. Discuss preoperative counseling in patients undergoing kyphoscoliosis correction surgery.

Ans. Explaining the wake-up test is most important. One must emphasize that though the patient may be awake, he/she will not feel pain neither he/she is likely to remember anything about it postoperatively. For pediatric patients, it is important to assure the parents, as the children are likely to be affected by the anxiety of the parents.

Q. Discuss methods to reduce blood loss and to avoid allogenic blood transfusion during kyphoscoliosis surgery.

Ans. Kyphoscoliosis surgery can be associated with major blood loss requiring blood transfusion. Therefore, every effort should be taken to reduce the blood loss.

Preoperative strategies:

- *Evaluation and treatment of hematological abnormalities:* Detailed history, examination, and laboratory testing should be done to identify and treat patients having bleeding tendencies or thrombotic episodes.
- *Evaluating risk factors for organ ischemia:* Risk factors for organ ischemia (ischemic heart disease and respiratory disease) should be identified and treated.
- *Evaluation and treatment of anemia:* Optimization of hemoglobin (Hb) preoperatively can reduce unnecessary blood transfusions. Hence, the cause of preoperative anemia should be identified and treated accordingly (preoperative oral or intravenous iron depending on the timing of the surgery, vitamin B₁₂, folic acid). Till date, evidence on perioperative erythropoietin (EPO) with or without iron therapy has been based on studies with significant heterogeneity related to dosing, timing of administration, severity of preoperative anemia, its cause, and type of surgery. There is no strong recommendation, but EPO can be administered possibly to reduce the need for allogeneic blood in a selected group of patients, e.g., renal insufficiency, anemia of chronic disease, and refusal of transfusion. Further

studies are needed to confirm its efficacy and safety in perioperative patients.

- **Drug history:** A detailed drug history of oral anti-coagulants, antiplatelets agents, and herbal medicines should be obtained. The risk of thrombosis versus bleeding should be evaluated and the latest guidelines should be followed while continuing or stopping anticoagulant and antiplatelet drugs. Changeover to short-acting anticoagulants such as bridging therapy with intravenous unfractionated heparin may be required in selected patients.
- **Preoperative autologous donation (PAD):** In well-scheduled surgeries, preoperative donation may be obtained if Hb is >11 g%. Subcutaneous EPO injections of 400 U/kg may be used to induce erythropoiesis 4 weeks before the scheduled date, and blood may be collected every 4–7 days starting 2 weeks before surgery, the last collection being done 3 days before surgery. High-risk of wastage of PAD units, need for meticulous planning several weeks before the intended date of surgery, and labor-intensive process are a few drawbacks of PAD because of which its use is slowly declining.

Intraoperative strategies:

- **Acute normovolemic hemodilution (ANH):** ANH may be considered in selected patients with normal initial Hb levels and are expected to lose two or more units of blood ($\geq 1,000$ mL) intraoperatively such as cardiac, thoracic, major orthopedic, and hepatic surgeries. In this technique, the patient's blood is removed shortly after induction of anesthesia and normovolemia is maintained by simultaneous replacement of volume by infusion of crystalloid and/or colloids as per institutional protocol. The target Hb is 7 g%. Starches should be used with caution in view of their effects on coagulation. The principle behind this technique is dilution of blood volume and production of deliberate, well-tolerated anemia so that the resultant blood loss is reduced during surgery. It is a safer technique when used in healthy, young adults with high risk for excessive bleeding and is a good option for Jehovah's Witnesses (if they accept the procedure). Meta-analyses of randomized controlled trials (RCTs) indicate that ANH is effective in reducing the volume of allogeneic blood transfusion for major cardiac, orthopedic, thoracic, or liver surgery. The advantages of ANH compared to PAD are transfusion of fresh blood to patients and its suitability in an unplanned surgery unlike PAD.
- **Proper positioning during surgery:** Probably, this is the most important factor, which has been discussed below with prone position.
- **Induced hypotension:** It should not be practiced as it makes the patient vulnerable to spinal ischemia, particularly if there are problems with positioning.

- **Normothermia:** Hypothermia is associated with impairment in platelet function and coagulation leading to increased blood loss. Therefore, normothermia should be maintained aggressively by means of forced air-warming devices.
- **Surgical technique:** Use of minimally invasive surgical techniques, use of electrocautery, and tissue adhesives can help to reduce surgical blood loss.
- **Intraoperative cell salvage:** Intraoperative blood salvage is retrieval and salvaging the blood that has already been shed during surgery. The shed blood is collected, washed, or filtered and returned to the patient, whenever transfusion is needed. Blood salvage is generally safe and is recommended in procedures with high blood loss ($\geq 1,000$ mL) or $\geq 20\%$ of the expected blood loss, low hemoglobin levels and high-risk of bleeding, rare blood group or multiple unexpected antibodies, and patient's refusal to receive allogeneic blood transfusion. It is associated with a very low incidence of adverse events and it reduces the frequency of allogeneic blood transfusions, particularly in major surgeries. Leukoreduction filters efficiently reduce the risk of contamination through amniotic fluid and fetal RBCs (red blood cells) in obstetric surgeries or in cancer surgeries; however, bowel contamination is considered as a contraindication for intraoperative blood salvage.

Q. What is transfusion trigger? What should be an ideal transfusion trigger?

Ans. Transfusion trigger is defined as that value of Hb below which RBC transfusion is indicated. Traditionally, the rule of "10/30" (Hb level 10 g/dL or a hematocrit 30%) has been followed for RBC transfusion. Due to variable transfusion practices, the exact transfusion trigger has not been defined. Although decisions to transfuse should be individualized on a case-to-case basis, overall "restrictive transfusion strategy" is widely accepted. Factors such as patient's age, comorbidities, ongoing bleeding or potential for further bleeding, and hemodynamic instability should be considered while making decision regarding blood transfusion, and broad guidelines should not surpass the clinical judgment in making the decisions regarding transfusion.

Clinical Practice Guideline 2016 from the American Association of Blood Banks (AABB) recommends a "restrictive transfusion strategy" with an Hb level of 8 g/dL as a transfusion threshold for patients undergoing orthopedic or cardiac surgery, and those with preexisting cardiovascular disease. However, the panel mentions that these recommendations are not applicable to patients with acute coronary syndrome, severe thrombocytopenia (patients treated for hematological or oncological reasons who are at risk of bleeding), and chronic transfusion-dependent anemia.

The following recommendations are made by American Society of Anesthesiologist's Task Force:

- Hb level is >10 g/dL: Transfusion is rarely indicated.
- Hb level is <6 g/dL: Transfusion is almost always indicated.
- Hb level is 6–10 g/dL: Transfusion should be based on any ongoing sign of organ ischemia or bleeding, the rate and magnitude of any potential or actual bleeding, and the patient's intravascular volume status.

Q. What is massive transfusion? What are the principles of management of massive transfusion?

Ans.

- Various definitions of massive blood transfusion (MBT) are available in the literature. The commonly used definitions are:
 - Replacement of one entire blood volume within 24 hours
 - Transfusion of >20 units of packed red blood cells (PRBCs) in 24 hours
 - Replacement of 50% of total blood volume (TBV) within 3 hours
 - Blood loss >150 mL/min with hemodynamic instability and further need for transfusion.
- Principles of management:
 - Maintaining and restoring intravascular volume: Compensatory mechanisms maintain organ perfusion till around 30% of TBV is lost. If adequate resuscitation is not started at this stage, organ hypoperfusion and shock can ensue. Hence, timely restoration of intravascular volume is critical.
 - Replacement of blood and blood components: Initial blood loss can be replaced with crystalloids or colloids. However, anemia and dilutional coagulopathy set in with increasing blood loss necessitating transfusion of blood components. Blood component therapy should be guided by laboratory testing. However, laboratory testing may cause delay in management as most of the tests take long time for their results. Hence, empiric treatment of ongoing loss with blood components based on institutional protocol is recommended alternatively.
 - Massive transfusion protocol (MTP): MTP is a strategy based on locally agreed guidelines for managing massive blood loss to help clinicians effectively manage massive blood loss, improve outcome, and minimize the adverse effects of hypovolemia and dilutional coagulopathy. Studies have shown that early administration of fresh frozen plasma (FFP) and higher ratios of FFP to PRBCs improve survival. The standard PRBC:FFP:platelet ratio suggested by MTPs is 1:1:1. Compliance to MTP has been shown

to be associated with decreased mortality. However, there are concerns with use of MTP as initiation of MTP is not standardized which may lead to wastage of blood components. Use of large amounts of blood products may also be associated with complications.

– Targets of resuscitation in massive blood loss:

- Mean arterial pressure (MAP) around 60 mm Hg, systolic arterial pressure 80–100 mm Hg (in hypertensive patients, one may need to target higher MAP)
- Hb 7–9 g/dL
- International normalized ratio (INR) <1.5 ; activated partial thromboplastin time (PTT) <42 seconds
- Fibrinogen >1.5 –2 g/L
- Platelets $>50 \times 10^9$ /L
- pH 7.35–7.45
- Core temperature $>35.0^\circ\text{C}$
- Lactates <2 mEq/L

Q. What are the complications of massive blood transfusion?

Ans. The complications of massive blood transfusion are given in Table 1.

TABLE 1: Complications of massive blood transfusion.

	Reaction	Mechanism
Acute reactions	Acute hemolytic transfusion reaction	• Incompatible transfused red cells • Patient's own anti-A or anti-B antibodies or other alloantibodies anti-rhesus (Rh) D to red cell antigens
	Nonhemolytic febrile reactions to transfusion of platelets and red cells	Due to patient antibodies to transfused white cells
	Transfusion associated dyspnea (TAD), transfusion-related acute lung injury (TRALI)	Due to donor plasma containing antibodies against the patient's leukocyte
	Mild-to-severe allergic reaction or anaphylaxis	Patient's antibodies that react with proteins in transfused blood components
	Transfusion-associated circulatory overload (TACO)	Rapid administration of blood products leading to pulmonary edema and acute respiratory failure
	Citrate toxicity	An anticoagulant present in significant amounts which readily binds calcium and magnesium resulting in hypocalcemia- and hypomagnesemia-associated cardiac manifestations

Contd...

Contd...

	Reaction	Mechanism
Delayed reactions	Delayed hemolysis of transfused cells	5–10 days post-transfusion, a rapid, secondary immune response raises the antibody level drastically, leading to the rapid destruction of transfused cells
	Alloimmunization	Development of anti-RhD in RhD-negative patients who have received RhD-positive cells which is fatal
	Development of antibodies that react with antigens of white cells or platelets	Development of leukocyte and/or platelet antibodies causing nonhemolytic febrile transfusion reactions in the future
	Post-transfusion purpura	Platelet-specific alloantibodies
	Graft versus host reactions	Caused by T-lymphocytes, they result in immunologic reaction against the immunodeficient host. It is devastating and fatal
	Iron overload	Each unit of blood contains 250 mg of iron and those receiving red cells over a long period may develop iron accumulation in cardiac and liver tissues
	Infection	Though rare but risk of HIV, hepatitis B, or hepatitis C from transfusion is present. Nonendemic infection such as human T-cell leukemia virus type 1 (HTLV-1) and type 2 (HTLV-2), West Nile virus, malaria and <i>Trypanosoma cruzi</i> (causing Chagas' disease), and variant Creutzfeldt–Jakob disease are also transmitted by BT
	Transfusion-related immunomodulation (TRIM)	Leukocytes are responsible for immunomodulatory effect

Q. What is your choice of anesthetic technique in this patient?

Ans. A combination of general and epidural anesthesia is the preferred anesthetic technique. Insertion of an epidural catheter (placed ideally before induction of anesthesia—this may not be possible in younger patients) requires a clear understanding of the rotation of spine. Epidural insertion may have medicolegal connotations, if there are residual neurological problems postoperatively. Anesthesia is induced with standard induction agents, either propofol

or thiopentone, in appropriate doses and neuromuscular blockade achieved with vecuronium 1 mg/kg or atracurium 0.5 mg/kg. Orotracheal intubation is performed with a proper-sized, nonkinking endotracheal tube. Eyelids are taped shut and eye pads are placed over them to cushion them in prone position.

Additional intravenous access (apart from that taken prior to induction) recommended for kyphoscoliosis correction is a 16 g cannula right forearm and a 20 g arrow central venous line via the right internal jugular vein. If acute isovolemic (or normovolemic) hemodilution is planned (if Hb is >12 g%, rare blood groups, etc.), one to two units of blood may be collected before the patient is positioned for surgery. Volume loss is replaced with colloids or Ringer lactate as per institutional protocol. An interpleural or paravertebral catheter may also be placed at this time to be used for postoperative analgesia.

Q. Discuss physiological changes in prone position.

Ans. Physiological changes associated with prone position are as follows:

Cardiovascular changes: Raised intrathoracic pressure causes reduction in venous return. The stroke volume is reduced with minimal changes in heart rates. Generally, the cardiac output has been shown to drop by about 20–30% of baseline value. MAP is maintained due to increased sympathetic activity (baroreceptor stimulation caused by reduced arterial filling).

Inferior vena cava obstruction is seen more often in patients where the abdomen is compressed (not hanging free). This compression can cause engorgement of the epidural spinal vessels leading to poor surgical field and increased blood loss.

Respiratory changes: Significant increases occur in functional residual capacity (FRC) and PaO_2 in prone position. In obese patients, lung volumes, compliance, and oxygenation have been found to be better than in supine position. Contrary to earlier belief, it must be emphasized that blood flow to dorsal regions is maintained even in prone position; thus, prone position is associated with a more uniform distribution of pulmonary blood flow and V/Q matching is improved in prone position.

Q. What monitoring will you employ? Discuss monitoring used for testing spinal integrity intraoperatively.

Ans. Standard monitoring employed includes electrocardiography, pulse oximetry, end-tidal CO_2 monitoring (most important to detect circuit disconnection, leaks during prone position), noninvasive and invasive blood pressure monitoring (with a 22 g cannula in the right radial artery), and urinary catheter for hourly monitoring of urine output. Airway pressure should be monitored again to detect kinking, displacement, and accidental removal of the endotracheal tube. Accidental extubation can have disastrous consequences.

Spinal integrity is monitored using either the traditional “wake-up test” or somatosensory evoked potentials (SSEPs).

Wake-up test: During this test, anesthesia is lightened intraoperatively and the patient is asked to perform certain commands. During kyphoscoliosis surgery, the patient may be asked to move or wriggle his/her toes, once it is confirmed that he/she is able to follow commands. Once the patient demonstrates the ability to perform these actions, the patient is put back to sleep. For this not to be a frightening experience for the patient, it is imperative that proper preoperative counseling is performed. The patient needs to be assured that though he/she may be awake, there will be no pain or discomfort. It is also important to ensure complete amnesia (with midazolam or some such agent), so that the patient does not have a postoperative recall of the test. A variety of techniques may be used for a prompt awakening intraoperatively. Total intravenous anesthesia (TIVA) with propofol and sufentanil or remifentanil and atracurium (with reversal for wake-up test) infusions is one such technique. A N_2O/O_2 /isoflurane, with switch to sevoflurane for the wake-up test, may also be used. The disadvantages of the wake-up test are an increased duration of surgery, a frightening experience for the patient, and the fact that the test provides information on neural damage only up to the time the test was performed. Damage occurring later is missed.

Evoked potential monitoring: An evoked potential is an electrical potential (at various loci such as cerebral cortex, brainstem, spinal cord, and peripheral nerves) after application of a stimulus, unlike the spontaneous potential detected by electroencephalogram. Various forms of stimuli can be applied and the response is recorded at different sites along the central nervous system. This can be of various types such as visual evoked potential (VEP), auditory evoked potential (AEP), SSEP, and motor evoked potential (MEP). During kyphoscoliosis surgery, SSEP has been most commonly used. Reports of transcranial (Tc) MEP are also present in the literature.

SSEP monitoring: It is used to monitor neuronal integrity during many surgeries (skull base surgeries which may require brainstem retraction, instrumentation of cervical spine, kyphoscoliosis correction surgery and surgeries for excision of spinal cord tumors, carotid endarterectomy, aneurysm repair surgery, and many other surgeries on various regions of spine), particularly spinal fusion procedures. An appropriate peripheral nerve is stimulated and the responses are recorded at the somatosensory cortex. Thus, a direct feedback is obtained testing posterior columns of the spinal cord. A change such as a decrease (>50%) in amplitude or an increase (>10%) in latency indicates an

interruption of the posterior column pathways. Though this sounds simple in theory, it requires particular skill and expertise as both amplitude and latency are affected by many anesthetic drugs (**Table 2**). Several other factors affect the SSEP. These are hypoxia, anemia, hypotension, hypothermia, nerve ischemia, and hypercapnia.

Advantage: SSEP provides continuous intraoperative monitoring of posterior column integrity during surgery, unlike the “wake-up test.”

Disadvantage: SSEP will not detect damage to the motor pathways. The effects of commonly used anesthetic agents on SSEPs are described in **Table 2**.

TABLE 2: Effects of anesthetic agents on somatosensory evoked potentials.

Anesthetic agents	Effect on amplitude	Effect on latency
Thiopentone	Small/none	Increased
Etomidate	Increased	Increased
Fentanyl	Small/none	Modest/no increase
Diazepam	Decreased	Increased
Midazolam	Decreased	Increased
Ketamine	Increased	Increased
Propofol	None	Increased
N_2O	Decreased	No change
Volatile agents	Decreased	Increased

MEP monitoring: MEP is recorded after either direct stimulation of the exposed cortex, or Tc cortical stimulation or magnetic cortical stimulation. TcMEP has been used to test neuronal integrity in kyphoscoliosis surgery.

In a large cohort of 500 patients undergoing surgical correction of idiopathic kyphoscoliosis, a combination of SSEP and TcMEP monitoring was used intraoperatively. The authors reported extremely small (0.014%, $n = 7$) false-positive rates, i.e., change in monitoring data without postoperative neurodeficit rates. The identification of postoperative neurodeficit was also excellent (two out of two). There were no false-negative results, i.e., postoperative neurodeficit without intraoperative adverse change in the monitoring data. The authors concluded that combined SSEP and neurogenic MEP monitoring during idiopathic scoliosis surgery represented a standard of care. It was felt that this combination obviated the need for an intraoperative wake-up test once reliable data were obtained and maintained.

Q. What are the risk factors for intraoperative spinal cord damage? How do you minimize spinal cord damage?

Ans. The risk for spinal cord damage is related to the length and type of surgical procedure, and pressure, retraction, and manipulation of the underlying neural tissue and

underlying spinal pathology. An intraoperative drop in spinal cord perfusion pressure will also lead to spinal cord damage. Hence, careful positioning and maintaining adequate mean arterial pressure (MAP) is important to ensure adequate spinal cord perfusion pressure [SCPP = MAP-ISP (intraspinous pressure)]. Studies in traumatic spinal cord damage suggest a doubtful neuroprotective role of methylprednisolone given within 8 hours of injury. Because a hematoma in the spinal canal can compress and produce spinal cord damage, careful hemostasis along with proper adjustment of anticoagulant medications perioperatively is extremely important.

Q. Discuss complications associated with prone position. Discuss postoperative visual loss seen after prone position.

Ans. Central nervous system complications of prone position are summarized in **Table 3**.

TABLE 3: Complication of prone position in the central nervous system.

Mechanism	Anatomy	Caused by	Clinical consequence
Arterial occlusion	Internal carotid artery, vertebral artery	Rotation, unrecognized cervical extension	Stroke, hemiplegia, quadriplegia, lateral medullary syndrome, brainstem and cerebellar infarcts
Venous occlusion	Abnormal veins	Poor positioning	Hemiparesis, paraparesis, quadriparesis bilateral venous infarcts
Air entrainment		Pneumorrhachis (air in spinal canal)	Transient quadriplegia
Cervical spine injury	Excessive neck flexion	Overstretching of cervical cord consequent ischemia	Transection below C5/6
	Excessive neck extension		Prolapsed intervertebral disk
Undiagnosed space-occupying lesions		Altered CSF flow dynamics epidural venous engorgement	Previously undiagnosed, lesions became symptomatic

(CSF: cerebrospinal fluid).

Peripheral nerve injuries have been described frequently (0.1–0.14%) after prone positioning to the following nerves: Supraorbital nerve (direct pressure during positioning), lingual and buccal nerves (inadvertent retraction of the jaw), brachial plexus, axillary nerve (both due to excessive

abduction of arms), musculocutaneous and radial nerves, ulnar nerve, recurrent laryngeal nerve and phrenic nerve (both caused by overextension or rotation of the neck), sciatic nerve, lateral cutaneous nerve of the thigh, and dorsal nerve of penis. Direct and indirect pressure during prone position can cause a variety of injury manifestations. These include contact dermatitis, pressure necrosis of skin at contact points, swelling of the tongue and oropharynx, compression of the trachea, swelling of the salivary glands, dislocation of the shoulder, mediastinal compression, visceral ischemia, and avascular necrosis of the head of femur. Limb compartment syndromes and rhabdomyolysis have also been reported after prone position.

Postoperative visual loss: It is a rare (0.0008%) but a dreaded complication of prone position. It has been reported that most (67%) cases of postoperative visual loss occur after prone positioning. A variety of ophthalmological injuries have been reported: Transient and permanent ophthalmoplegia, orbital compartment syndrome, dislocation of intraocular lens, etc. The most commonly described injuries are ischemic optic neuropathy and central retinal artery occlusion. These two are compared in **Table 4**.

TABLE 4: Comparison of ischemic optic neuropathy and central retinal artery occlusion.

	Ischemic optic neuropathy	Central retinal artery occlusion
Etiology	Intraoperative hypotension, prolonged surgery, excessive blood loss, large amounts of crystalloid infusion	Direct external pressure emboli
Mechanism	- Ischemia (due to the above factors) - Orbital edema leading to stretching/compression of the optic nerve	Decreased ocular perfusion pressure
Clinical features	Painless, bilateral, decreased light perception and visual fields	Painless, unilateral, periorbital swelling or ecchymosis

Q. Discuss postoperative analgesia.

Ans. Postoperative pain control following spinal fusion is usually based on patient (or nurse) controlled analgesia (PCA or NCA) with opioids, supplemented with either nonsteroidal anti-inflammatory agents (for the pain at the site of surgery) or benzodiazepines (for muscle spasms). Children with cerebral palsy are vulnerable to undertreatment of postoperative pain due to communication barriers, concerns regarding opioid-induced respiratory depression, and individual opinion about pain perception in these children. Children with cognitive impairment, for whom the behavioral pain scale was usually used, received

smaller total opioid dose than those without cognitive impairment, for whom self-report was used. There is mounting evidence that epidural morphine is safe and efficient for postoperative analgesia following spine surgery. Low-dose (2–5 µg/kg) intrathecal morphine administered in the operating room supplemented by PCA morphine provided better analgesia than PCA morphine alone, especially at rest; pain scores at coughing were similar in the morphine and placebo groups. Similarly, patients who received intrathecal morphine and sufentanil for surgery were breathing at a normal rate after surgery except for the first postoperative hour, when PaCO₂ was slightly elevated. No late respiratory depression was observed, but continuous monitoring of ventilation was considered necessary for at least 24 hours. There is no consensus in the literature regarding the benefits of epidural catheters after spinal surgery.

Clinical experience has indicated that the use of a continuous epidural infusion of local anesthetics with morphine delivered through a single epidural catheter provided improved analgesia when compared with intravenous opioids. However, it was noted that many patients still had significant pain in the upper or lower portion of the surgical site. Tobias and coworkers reported placing two catheters following completion of the surgical procedure and prior to wound closure. One of them was inserted at the level T6-8, with the tip directed cephalad, to T1-4, and the other was inserted at T12 and positioned at the L1-4 levels. As the surgical wound was being closed, fentanyl and hydromorphone were administered through the catheters. This was followed by continuous infusion of 0.1% ropivacaine and hydromorphone 10 mg/mL. The mean of the median pain scores on the postoperative days 1–5 was always <2 and the mean of the maximum pain scores was <4 on the postoperative days 1–5. Although the technique was effective, many of the patients still had episodes of severe pain with maximum pain scores of 6–7. No adverse effects related to epidural analgesia were noted. To avoid the possible additive respiratory-depressant effects of the intravenous and epidural opioids, Tobias and coworkers used a short-acting opioid (remifentanyl) during the surgery. Interpleural catheters or epidural catheters can also be very useful for pain relief.

Q. What are the complications associated with scoliosis surgery?

Ans. Aside from the complications due to intraoperative compromise of spinal integrity, which may give rise

to variable neurological deficits, the complications of corrective surgery for scoliosis may be related to massive blood loss, neural deficit due to prone positioning, and postoperative visual loss. The greater the degree of scoliosis, particularly above 100°, the higher the chance of postoperative complications. The rate of complications is also greater in patients with cerebral palsy, thoracoabdominal and transthoracic approaches, staged procedures, operative blood loss >1,000 mL, or previous spine surgery. Pulmonary complications are also a frequent result of scoliosis surgery. Other complications of scoliosis surgery include syndrome of inappropriate antidiuretic hormone release, pancreatitis, cholelithiasis, superior mesenteric artery syndrome, ileus, chylothorax, air and fat embolism, and tension pneumothorax.

SUGGESTED READING

1. Chandanwale AS, Ramteke AA, Barhate S. Intraoperative somatosensory-evoked potential monitoring. *J Orthop Surg. (Hong Kong)* 2008;16:277-80.
2. Crabb I. Anesthesia for spinal surgery. *Anaesth Intensive Care Med.* 2003;80-4.
3. Edgcombe H, Carter K, Yarrow S. Anesthesia in the prone position. *Br J Anaesth.* 2008;100:165-83.
4. Gambrall MA. Anesthetic implications of corrective surgery for kyphoscoliosis. *AANA J.* 2007;75:277-85.
5. Khasne RW, Kulkarni AP, Kulkarni PA. Perioperative blood transfusion. In: Kulkarni AP, Divatia JV, Solanki S (Eds). *Perioperative Critical Care.* Jaypee Brothers Medical Publishers: New Delhi. (In Press).
6. Padberg AM, Wilson-Holden TJ, Lenke LG, Bridwell KH. Somatosensory and motor evoked potential monitoring without a wake-up test during idiopathic scoliosis surgery. An accepted standard of care. *Spine* 1998;15:1392-400.
7. Patil V, Shetmahajan M. Massive transfusion and massive transfusion protocol. *Indian J Anaesth.* 2014;58(5):590-95.
8. Penney R. Use of dexmedetomidine and ketamine infusions during scoliosis repair surgery with somatosensory and motor-evoked potential monitoring: a case report. *AANA J.* 2010;78:446-50.
9. Raw DA, Beattie JK, Hunter JM. Anesthesia for spinal surgery in adults. *Br J Anaesth.* 2003;91:886-904.
10. Roth S. Perioperative visual loss: what do we know, what can we do? *Br J Anaesth.* 2009;103S(1):i31-40.
11. Thuet ED, Winscher JC, Padberg AM, Bridwell KH, Lenke LG, Dobbs MB, et al. Validity and reliability of intraoperative monitoring in pediatric spinal deformity surgery: a 23-year experience of 3436 surgical cases. *Spine (Phila Pa 1976).* 2010;35:1880-6.
12. Vitale MG, Moore DW, Matsumoto H, Emerson RG, Booker WA, Gomez JA, et al. Risk factors for spinal cord injury during surgery for spinal deformity. *J Bone Joint Surg Am.* 2010;92:64-71.

Large Thyroid Mass

Madhavi Shetmahajan, Raghu S Thota, Bindiya Salunke

A 54-year-old female patient presents with a large thyroid swelling. She has no comorbidities. She is scheduled to undergo thyroidectomy. We discuss the perioperative anesthesia management.

The focus of this case is airway management and management of hypo-/hyperthyroidism.

Q. What specific things are you seeking in the history and examination of a patient with thyroid swelling?

Ans.

Size and duration of swelling:

- *Pressure effects on the trachea:* Dyspnea, stridor (and effects of change in position)
- *Pressure effects on the esophagus:* Dysphagia
- *Pressure effects on the recurrent laryngeal nerve (RLN):* Hoarseness
- *Duration:* Long-standing large thyroid may be associated with tracheomalacia.

Retrosternal extension:

- Dysphagia, dyspnea
- Unable to "get under the swelling"
- Superior vena cava (SVC) obstruction/thrombosis—dilated veins in the neck and upper part of the chest, facial edema.

Intratracheal extension:

- Dyspnea
- Hemoptysis.

Thyroid hormone status:

- Signs and symptoms of hypo- or hyperthyroidism.

Associated endocrine disorders:

- *Multiple endocrine neoplasia (MEN) type 2A:* Medullary thyroid carcinoma, pheochromocytoma, parathyroid hyperplasia
- *MEN type 2B:* Medullary thyroid cancer, pheochromocytoma, and growths around nerves (neuromas)
- *MEN I:* Pituitary adenoma, parathyroid hyperplasia, pancreatic tumor-like insulinoma, and rarely thyroid and adrenal involvement.

Thyroid malignancies:

- Usually present as thyroid nodules and are usually metabolically inactive
- *Common types:* Papillary, follicular, medullary, anaplastic
- Intratracheal extension may present as dyspnea and hemoptysis
- Distant metastasis to the lung, bones, and liver may be present, especially in the follicular type.

Q. How would you present your diagnosis?

Ans. A 54-year-old female with size/side (e.g., large thyroid swelling/4 × 3 cm swelling on right) with/without airway compromise (e.g., with tracheal deviation and compression) with/without complications (e.g., retrosternal extension without SVC syndrome or compression of esophagus/RLN) with normal thyroid function (or controlled/uncontrolled hypothyroidism/hyperthyroidism) with/without comorbidities (e.g., well-controlled diabetes mellitus) is scheduled for subtotal thyroidectomy.

Q. How are thyroid hormones synthesized in the body?

Ans. In the thyroid gland, active uptake of iodide occurs in exchange for Na⁺. Iodide uptake is stimulated by thyroid-stimulating hormone (TSH). Iodide is oxidized to active iodine by hydrogen peroxide (H₂O₂). The reaction is catalyzed by thyroid peroxidase (TPO). Iodine is actively transported across the apical surface of the follicular cell. Active iodine is incorporated into the tyrosine residues of thyroglobulin molecules to form mono- and diiodotyrosines (MIT and DIT).

Thyroglobulin is taken up into the colloid of the follicle where a coupling reaction between pairs of iodinated tyrosine molecules occurs. The coupling of two DIT

produces tetraiodothyronine or thyroxine (T_4) while the combination of DIT with MIT produces triiodothyronine (T_3). This reaction is catalyzed by TPO. Thyroid hormones are stored in this state and are only released when the thyroglobulin molecule is taken back up into the follicular cells. Thyroglobulin droplets are taken up by the follicular cells by the process of pinocytosis which is stimulated by TSH. Fusion of the droplets with lysosomes results in hydrolysis of the thyroglobulin molecules and the release of T_4 and T_3 . Approximately 100 μg of thyroid hormones are secreted from the gland each day, with around 90% in the form of T_4 and the remainder as T_3 . T_4 undergoes peripheral conversion to T_3 (T_3 is three to five times more active than T_4) in the liver and kidney or to reverse T_3 (little or no biological activity). Glucocorticoids, propranolol, and amiodarone block conversion of T_4 to T_3 .

About 99.98% of T_4 and 99.8% of T_3 is bound. It is the free fraction which is responsible for the actions and the negative feedback to the pituitary gland.

T_3 and T_4 regulation: The production of T_4 and T_3 is regulated by TSH released by the anterior pituitary, which is in turn released as a result of thyrotropin-releasing hormone (TRH) released by the hypothalamus. The thyroid and thyrotropes form a negative feedback loop. TSH production is suppressed when the free T_4 levels are high and vice versa.

Q. What are the actions of thyroid hormone?

Ans. Thyroid hormones act on almost all the cells of the body with effects on growth and development. Many of the actions of thyroid hormones are mediated by their binding to nuclear receptors that have a preferential affinity for T_3 . T_3 receptors are steroid hormone receptors which regulate the expression of genes coding for enzymes which regulate cell function.

Central nervous system (CNS): Effect on neuronal function and reflexes. The reaction time of stretch reflex is shortened in hyperthyroidism. The thyroid hormone also affects the reticular activating system.

Cardiovascular system (CVS): Actions of thyroid hormones and catecholamines are interrelated. Thyroid hormones have actions similar to epinephrine in stimulating the cardiovascular system, nervous system, and metabolic rate. Thyroid hormones increase the number and affinity of β -adrenergic receptors, thus increasing their sensitivity to catecholamines causing increased cardiac output and tachycardia.

Metabolism: Thyroid hormones increase the metabolic rate and oxygen consumption of most tissues. They also increase carbohydrate absorption from the gut, increase protein catabolism, mobilize fatty acids, and lower serum cholesterol.

Growth: Thyroid hormones are important for brain development and skeletal maturation.

Q. How will you clinically assess the thyroid function status in the patient? What are the signs and symptoms of hypo- and hyperthyroidism? State eye signs in hyperthyroidism.

Ans. History of taking any medicines—duration and dose, previous thyroid function test (TFT).

Symptoms and Signs of Hyperthyroidism

Hyperactivity, weight loss, and tremor (tested in outstretched hands and tongue) are classical symptoms. Other findings are warm moist skin, diarrhea, intolerance to heat, weakness in large muscle groups, menstrual abnormalities, and osteopenia.

Common cardiac findings are tachycardia (especially a high sleeping pulse rate), dysrhythmia (atrial fibrillation is common), mitral valve prolapse, heart failure, and ischemic heart disease (IHD).

Eye signs: Eyelid retraction ("stare") (Dalrymple sign): The eyelids are retracted upward above the superior corneoscleral limbus and the "white" of the sclera is seen.

Lid-lag (von Graefe's sign): When the patient tracks an object downward with his/her eyes, the eyelid fails to follow the downward moving iris, and the same type of upper globe exposure which is seen with lid retraction is seen temporarily.

Joffroy's sign: Absence of wrinkling of the forehead when looking upward with the face tilted downward. These ocular signs are not the same as exophthalmos (protrusion of the eyeball) which occurs specifically in Graves' disease. The forward protrusion of the eyes is due to immune-mediated inflammation in the retro-orbital fat. Exophthalmos may exacerbate hyperthyroid lid-lag and stare.

Thyroid function tests:

- Low TSH, high free T_3 , T_4
- Subnormal TSH response to TRH
- **Other blood tests:** Mild anemia, thrombocytopenia, hypercalcemia, raised alkaline phosphatase.

Symptoms and Signs of Hypothyroidism

Cardiovascular system: Bradycardia, abnormal baroreceptor function, reduced plasma volume, signs of heart failure, pericardial effusion.

Respiratory system (RS): Impaired respiratory center control mechanisms, respiratory muscle weakness, pleural effusions.

Central nervous system: Slow movement, mental slowing, excessive sleepiness, depressed mood.

Gastrointestinal system: Delayed gastric emptying, constipation. Impaired clearance of free water and impaired hepatic drug metabolism:

- Fatigue, modest weight gain, cold intolerance, menorrhagia, muscle cramps, vague aches, and pain
- Dry coarse hair, dry skin, large tongue, swelling of the legs
- Anemia, hypoglycemia, hyponatremia, increased cholesterol levels
- Carpal tunnel syndrome.

Q. How will you test thyroid function?

Ans. Total T_4 , T_3 , and TSH are the basic tests for diagnosis of hypothyroidism and hyperthyroidism (Table 1).

Free T_4 and T_3 : These are better indicators of the functional thyroid status than total levels as they are the metabolically active fractions and are not affected by thyroxine-binding globulin (TBG) levels. The relationship between total and free levels depends upon binding to TBG which is affected by various conditions. TBG is increased by estrogens, tamoxifen, and pregnancy and in some cases of liver disease, including hepatitis. Here, there would be a high total value but normal function. Decreased TBG is found with some cases of chronic liver disease, nephrosis, acromegaly, severely ill patients, and patients on large doses of glucocorticoids and androgens/anabolic steroids.

Thyroid-stimulating hormone: It is an early indicator of hypo and hyperthyroidism in the presence of a normal pituitary thyroid axis and is a useful screening test. TSH is not useful when the pituitary thyroid axis may be disturbed such as at initiation of thyroxine therapy, pregnancy, and in patients with hyperthyroidism in the early stage of treatment and in rare situations such as central hypothyroidism, end-organ thyroid hormone resistance, and TSH-secreting pituitary adenomas.

TABLE 1: Diagnosis of hyperthyroidism and hypothyroidism.

	Hyperthyroidism	Hypothyroidism	
		Primary	Secondary
TSH	Low	High	Low
T_4	High	Low	Low

Other advanced investigations include TBG level, radioiodine uptake test, and antithyroglobulin antibodies. TFTs are advisable in patients with goiter to detect subclinical hypo- or hyperthyroidism.

Q. How is subclinical hypothyroidism (SHypo) and subclinical hyperthyroidism (SHyper) managed?

Ans. The subclinical condition is diagnosed when TSH levels are abnormal in the presence of normal free triiodothyronine 3 (FT_3) and FT_4 .

SHypo treatment (2013 European Thyroid Association Guidelines):

- **TSH > 10 mU/L:** Age < 70 years—treated with levothyroxine, age > 70 years—symptomatic patients should be considered for treatment.
- **TSH 5–10 mU/L:** Age < 70 years—symptomatic patients should be given a trial of 3 months to look for a response. All other patients with TSH > 5 mU/L should get a TFT done in 6 months to look for progress.

SHyper treatment (2015 European Thyroid Association Guidelines):

- **TSH 0.1–0.39 mU/L:** Follow up at 3–6 months. Patients with persistent SHyper may require treatment with either antithyroid drugs, β -blockers, or radioiodine therapy based on risk factors.
- **TSH < 0.1 mU/L:** All patients need further evaluation and most would need treatment.

Q. What is euthyroid sick syndrome?

Ans. The euthyroid sick syndrome is described as abnormal TFTs that occur in the setting of acute and severe nonthyroidal illness without preexisting hypothalamic pituitary and thyroid gland dysfunction. The most common findings are low T_3 , T_4 , and TSH. The TFT abnormalities are reversible after recovery from the illness.

These changes may be caused partly by cytokines or other inflammatory mediators acting at the hypothalamus, pituitary, thyroid gland, and hepatic deiodinase system. The degree of thyroid function disturbance generally correlates with disease severity. It is not certain whether these changes are part of an adaptive response to lower tissue energy requirements during disease or could be a maladaptive response. Administration of thyroid hormones in this situation is controversial and has not been shown to improve outcomes.

Q. How important is it to treat hyperthyroidism/hypothyroidism?

Ans. The thyroid hormone is highly metabolically active, has vital effects on cardiovascular physiology, and affects the response to anesthetic drugs. The stress of surgery and anesthesia can lead to decompensation of both hypothyroid and hyperthyroid states increasing perioperative morbidity and mortality. Also, manipulation of the thyroid gland during surgery may cause the release of thyroid hormone and precipitate a hyperthyroid crisis. So, the endocrine status should be medically controlled and patients rendered euthyroid prior to surgery.

Q. What are the causes of hyperthyroidism?

Ans. Graves' disease, thyroiditis, toxic multinodular goiter, toxic adenomas, iodine-induced, struma ovarii, thyroxine

overdose, side effects of certain medications such as amiodarone, irradiation thyroiditis, etc.

Q. How is hyperthyroidism treated?

Ans. Hyperthyroidism can be treated by antithyroid medications, surgery, and radioactive iodine 131 therapy.

The treatment has to be individualized to each patient. Antithyroid drugs, namely carbimazole, methimazole, and propylthiouracil (PTU), act by interfering with the organification of iodine. Carbimazole acts by its conversion to methimazole in the liver. PTU in large doses also blocks the peripheral conversion of T_4 to T_3 . Antithyroid drugs are usually the first-line treatment and can be continued or followed by surgery or radioactive iodine in suitable cases.

The initial oral dose of methimazole is 10–30 mg/day and that of carbimazole is 15–40 mg/day. The PTU dose is 100 mg every 8 hours. The doses are titrated to response. When initiating treatment, the patient is investigated 3–4 weekly for free T_3 and T_4 levels till control is achieved. TSH is a poor indicator of response at the initiation of treatment. The maintenance dose is usually 2.5–10 mg of methimazole or 50–100 mg of PTU daily.

β -blockers are used in early treatment for control of palpitations and tremors. Propranolol 20–40 mg 6 hourly is commonly used. Longer acting β -blockers, e.g., atenolol or bisoprolol, may also be used. High doses of propranolol also have an added advantage of inhibiting the peripheral conversion of T_4 to T_3 . Cardioselective β -blockers may be preferred in asthmatics and in those at risk for atrial fibrillation.

Patients with atrial fibrillation require anticoagulation. It should be noted that patients with hyperthyroidism require higher doses of digoxin.

Patients with Graves' disease in remission need to be investigated for thyroid hormone levels as relapses are known.

Q. What are the common side effects of antithyroid drugs?

Ans. Major side effects of antithyroid drugs (so corresponding investigations should be ordered preoperatively):

Agranulocytosis: May need granulocyte colony-stimulating factor.

Hepatotoxicity: Immunoallergic hepatitis seen with PTU.

Vasculitis: Acute renal dysfunction, arthritis, skin ulcerations, vasculitic rash, and upper and lower respiratory symptoms, including sinusitis and hemoptysis. May need high-dose glucocorticoid therapy or cyclophosphamide.

Teratogenicity: PTU better than methimazole during pregnancy. Both drugs are safe for lactation.

Iodides: Iodides inhibit hormone release at high concentration, decrease synthesis, and block the conversion

of T_4 to T_3 . The onset of action is immediate but lasts only for a few weeks. Three drops of a saturated solution of potassium iodide are given 8 hourly or the radiocontrast dye, ipodate, can be given 0.5–3 g every day.

Q. Describe anesthesia management in a hyperthyroid patient.

Ans. Surgery is delayed until the patient is euthyroid. An endocrinologist consultation is required to titrate therapy. Only life-/limb-/organ-threatening emergency surgery should be done in an uncontrolled hyperthyroid state. (Sight-threatening Graves' ophthalmopathy is an endocrine emergency. The patient may require orbital decompression before the hyperthyroid state is completely controlled).

Elective surgery: Elective surgery should be undertaken after a minimum of 3–4 weeks of therapy with methimazole or PTU. The delay is essential to achieve control as there are large stores of thyroid hormone. Free T_3 and T_4 are recommended for evaluating the control of the hyperthyroid state. Change in TSH levels is slower; therefore, TSH may not be representative of an early response to treatment.

If surgery is semiurgent, it should be scheduled after 7–14 days. Treatment with iodide and propranolol is advisable. Antithyroid drugs should also be started even though they by themselves would not achieve control. Dexamethasone 2 mg 6 hourly intravenous (IV) reduces hormone release as well as inhibits the peripheral conversion of T_4 to T_3 . Lugol's iodine given for 10 days preoperatively in a dose of 0.5 mL three times a day may be useful to reduce the vascularity of the thyroid gland and decrease surgical blood loss as antithyroid drugs make the thyroid more vascular. Although this practice is no longer routinely followed before thyroid surgery, it is finding favor for early control of the hyperthyroid state. It is important that patients are started on antithyroid drugs before iodides are started as the administration of iodides in iodine-deficiency patients may worsen the hyperthyroid state. Vitamin D deficiency should be corrected prior to surgery to reduce the risk of postoperative hypocalcemia.

Emergency surgery: Esmolol is titrated to control heart rate [caution in patients with congestive heart failure (CHF)]. It is administered in the dose of 0.5 mg/kg IV followed by an infusion of 0.03–0.3 mg/kg/min.

Anesthesia management for thyroidectomy in a patient with hyperthyroidism: General anesthesia is the technique of choice for thyroidectomy. Some centers specialize in thyroid surgery under cervical plexus blocks or cervical epidural anesthesia. These should only be attempted by anesthetists skilled in the procedure for a select group of patients.

All antithyroid medications are continued on the morning of surgery. Sedative premedication is prescribed if the airway is not involved. Benzodiazepines reduce anxiety and catecholamine release. Anticholinergic drugs cause tachycardia and alter heat-regulating mechanisms and are best avoided.

Monitoring would include SpO₂, BP, electrocardiography (ECG), EtCO₂, and temperature. Arterial cannula and continuous BP monitoring are indicated in patients with an uncontrolled thyroid condition.

Maintain a deep plane of anesthesia so as to reduce the sympathetic response to surgical stimulation. Hyperthyroidism has not been seen to increase minimum alveolar concentration (MAC) in animal studies. Isoflurane and sevoflurane are inhalational agents of choice.

Thiopentone decreases peripheral conversion of T₄ to T₃ and is preferred. Ketamine is avoided due to sympathomimetic action. Clearance and distribution volume of propofol are increased in hyperthyroid patients. When total IV anesthesia (TIVA) is used, propofol infusion rates should be increased.

Besides pancuronium which has a vagolytic action, muscle relaxants in current practice, e.g., vecuronium or rocuronium, have minimal hemodynamic side effects and are safe. Succinylcholine may be used in indicated cases.

Avoid indirect-acting vasopressors, e.g., ephedrine. Phenylephrine, a directly acting alpha-adrenergic receptor agonist, is the drug of choice for hypotension. Avoid adrenaline-containing solutions. Glycopyrrolate is used with neostigmine for reversal.

Antithyroid drugs are stopped after thyroidectomy but β -blockers are continued for 7–10 days as the half-life of thyroxine is 7–8 days.

Q. What is thyroid steal?

Ans. "Thyroid steal" is a historical practice used by George W Crile in the early 1900s for patients planned for thyroidectomy for Graves' disease. The aim was to avoid preoperative stress associated with the anticipation of surgery which could lead to thyroid storm. The patients were admitted without knowing the day of surgery. Every morning, saline injections were given to the patient which were substituted with anesthetic drugs on the day of surgery. Thus, in a way, as the patient was not actually aware that he/she was to be operated that day, the thyroid was "stolen." *This is not to be confused with the "thyroid steal syndrome" where blood is stolen (diverted) from cerebral circulation to thyroid circulation due to excessive vascularity of thyroid gland or nodule, causing transient cerebral ischemia.*

Q. What is a thyroid storm?

Ans. It is a life-threatening condition where exacerbation of the hyperthyroid state is precipitated by acute stress such as infection, surgery, and trauma. It has been reported up to 12–18 hours after surgery in hyperthyroid patients. Patients on amiodarone can sometimes develop this condition. Tachycardia and hyperthermia are classical signs. Other diagnostic criteria for thyroid storm in Graves' disease include arrhythmias, CHF, agitation, delirium, psychosis, stupor, coma, nausea, vomiting, diarrhea, and hepatic failure.

Thyroid function tests are not diagnostic because the condition is thought to be precipitated by decreased binding of thyroid hormones to TBG increasing the amount of the free fraction.

Differential diagnosis: Malignant hyperthermia, neuroleptic malignant syndrome, pheochromocytoma.

Treatment: Treat the precipitating cause, aggressive fluid management, cooling, PTU, sodium iodide, and methylprednisolone 50 mg IV.

α - and β -blockers should be given under intensive hemodynamic monitoring. Magnesium has also been used to control dysrhythmias.

Q. What are the causes of hypothyroidism?

Ans. Hashimoto's thyroiditis, lymphocytic thyroiditis, thyroid destruction (from radioactive iodine or surgery), pituitary or hypothalamic disease, radiation therapy to the neck for nonthyroid malignancies, medications, e.g., tyrosine kinase inhibitors, and severe iodine deficiency.

Some genetic disorders present as multiple autoimmune endocrinopathies (MAEs). They are subdivided as type 1 MAE and type 2 MAE on the basis of the following characteristics:

- *Type 1 MAE:* Hypoparathyroidism, Addison's disease, and mucocutaneous candidiasis. Autoimmune thyroiditis is present in about 10–15% of patients.
- *Type 2 MAE:* Addison's disease, autoimmune thyroiditis, and type 1 diabetes.

Q. What is myxedema coma?

Ans. Myxedema coma is a loss of brain function as a result of severe, long-standing hypothyroidism. This condition is mostly seen in elderly patients and tends to occur more often in women. The condition carries a very high mortality.

Factors which may trigger myxedema coma in a person with poorly controlled hypothyroidism include drugs (particularly sedatives, narcotics, anesthesia, lithium, and amiodarone), infections, stroke, trauma, heart failure,

gastrointestinal bleeding, hypothermia, and failing to take thyroid medications as prescribed.

Treatment: The combination of IV T_3 and T_4 is recommended. Fluid resuscitation is done with a normal saline solution. Glucose supplementation is given as required. Normothermia should be achieved with active warming. Mechanical ventilation and vasopressors may be required.

Q. How will you treat hypothyroidism?

Ans. L-thyroxine: Treatment is started with 50–100 μg (25 μg in the elderly or in patients with IHD). The dose is titrated to clinical improvement and by monitoring TSH levels. Estimation of TSH and free T_4 is recommended for evaluating control.

T_4 has a half-life of 7 days. The onset of action is at 12 hours and it takes almost 2 weeks for peak action. The half-life of T_3 is 1.5 days and is available in an injectable form.

Thyroxine supplementation may precipitate or aggravate IHD. In patients with significant IHD, early revascularization and immediate postoperative initiation of thyroxine supplementation are advisable.

Q. Discuss the implications of hypothyroidism on fitness for anesthesia and surgery.

Ans. Patients with overt hypothyroidism should be treated prior to elective surgery. Normalization of thyroid status will typically take weeks. If surgery is urgent but can be delayed for 24–48 hours, IV T_3 can be given for quicker action. The peak action of T_3 occurs at 36–72 hours. Because of an autoimmune etiology, both adrenocortical insufficiency and myasthenia gravis are more common in hypothyroid patients. Due to reduced adrenocorticotrophic hormone response to stress, hypothyroid patients should receive hydrocortisone cover during periods of increased surgical stress. Hypothyroid patients should receive their usual morning dose of thyroxine. This is especially important for T_3 which has a shorter half-life.

Mild or subclinical hypothyroidism is unlikely to affect the perioperative course; therefore, postponement of surgery is not generally advocated.

Q. Describe anesthesia in a hypothyroid patient (emergency surgery).

Ans. The risk of complications is higher when patients have an $\text{FT}_4 < 0.5 \mu\text{g/dL}$ or $\text{T}_4 < 1 \mu\text{g/dL}$.

Use regional anesthesia wherever possible.

Specific investigations:

- Hemoglobin count as anemia is common
- Platelet count and clotting tests as dysfunction is known
- Serum electrolytes as hyponatremia can be present
- Blood sugar monitoring to detect hypoglycemia
- ECG—bradycardia, conduction blocks.

Avoid sedative premedication in overtly hypothyroid patients as they show increased sensitivity to sedative and

anesthetic drugs, probably related to low cardiac output, reduced blood volume, impaired hepatic metabolism, and abnormal baroreceptor function. Chemoreceptor responses are also blunted, increasing the risk of respiratory depression. Close postoperative monitoring is advisable.

Induction: Anesthetic drugs have to be used judiciously. Ketamine has the advantage of sympathetic stimulation. Fentanyl may be used in small titrated doses. Nitrous oxide is useful in reducing the requirement of volatile anesthetics as these patients are sensitive to the myocardial depressant action of the latter. Intermediate-duration nondepolarizing agents, e.g., vecuronium, are used. Pancuronium is not preferred in spite of its cardiovascular effects because of the risk of prolongation of action and residual neuromuscular blockade (NMB).

Exaggerated hypotension is common and should be treated with judicious fluid and ephedrine. Pure alpha-adrenoreceptor agonists are avoided. Dopamine and epinephrine can be used for severe hypotension. Such patients should also receive an additional dose of steroids.

Patients may have airway edema, large tongue, and enlarged thyroid making laryngoscopy and intubation difficult.

Intraoperatively monitor temperature, fluid and electrolyte status, and blood sugars. Invasive BP monitoring ($\pm$ central venous cannulation and cardiac output monitoring) may be required in hypothyroid patients undergoing major surgery (fluid shifts, blood loss, etc.) and in those with cardiovascular involvement (arterial cannula under local anesthesia prior to induction is advisable). Neuromuscular monitoring with a peripheral nerve stimulator is advised as these patients have increased the incidence of myasthenia gravis. Higher current levels may be needed for nerve stimulation in hypothyroid patients due to thick skin and hypothermia. Warming measures are instituted to prevent hypothermia. Delayed recovery is common and postoperative ventilation may be required.

Q. What problems do you anticipate in patients with large thyroid mass? What are the complications associated with retrosternal thyroid?

Ans.

- Tracheal compression, deviation, invasion. Tracheomalacia may be present in long-standing cases which can predispose to intraoperative and postoperative airway collapse.
- Worsening of airway compromise in the supine position and with loss of muscle tone. Loss of spontaneous breathing and negative intrathoracic pressure after anesthesia and paralysis can also precipitate the collapse of the intrathoracic airway.
- **Retrosternal extension:** This can lead to SVC syndrome, airway compromise, cardiac compression, cerebral hypo-perfusion as a result of arterial compression

and thyrocervical steal, phrenic and RLN palsies, Horner's syndrome, pleural effusions, chylothorax, and pericardial effusions.

- Increased intraoperative bleeding.
- Injury to laryngeal nerves—vocal cord palsy causing stridor and impaired airway reflexes, thereby increasing the aspiration risk.

Q. How will you assess for tracheal deviation and compression?

Ans. Patients with tracheal narrowing by >30% are usually symptomatic and the risk of airway events increases when >50% narrowing is present. Patients with malignant tumors may have lymph node masses which may exert pressure effects on the surrounding structures.

History: Difficulty in breathing, stridor, or stridor on lying down.

Examination:

- *For deviation:* Palpation of and auscultation on the anterior neck to diagnose deviation
- *For compression:* Positive Kocher's test: Slight push on the lateral lobes causes stridor.

Investigations:

Computed tomography (CT) scan of the neck and upper thorax: Patients with significant mass effects or malignant tumors are likely to undergo a CT scan as part of the surgical workup. If not, it should be advised by an anesthetist in case of suspected airway compromise.

This helps to accurately delineate the site and degree of airway compromise. This also helps to predict the tracheal tube diameter and guide placement. A note of caution when assessing tracheal dimensions on CT scan: The tracheal cuts are slightly oblique on a standard CT image and may overestimate the tracheal diameter.

CT scans performed in different positions may be useful to record positional changes in the airway compression.

X-ray neck: Anteroposterior (AP) and lateral. With the widespread availability of CT scans, X-ray imaging of the trachea has a very limited role in current practice. It may be useful to detect tracheal compression and deviation in a resource-poor environment.

Flow volume loops: These may help in diagnosing subclinical airway obstruction, particularly dynamic obstruction. Flow volume loops will not change management in the presence of overt obstruction. Their practical role in airway assessment in an asymptomatic thyroid patient is debatable. The pattern seen on the flow volume loop depends on the site of tracheal compression (if it is a variable obstruction). Suprasternal compression will show an extrathoracic pattern, i.e., the inspiratory flow is predominantly

affected whereas infrasternal compression will have an intrathoracic pattern, i.e., expiratory flow is affected. Large goiters and intratracheal extensions usually cause fixed obstruction where both inspiratory and expiratory flows are compromised.

Q. What are the problems associated with intratracheal extension of the tumor?

Ans. These patients have a high risk of bleeding and tumor dislodgement leading to critical airway obstruction. Intubation should be attempted after a thorough examination of the airway including CT scan and flexible bronchoscopy. Rigid bronchoscopy with appropriate instruments and efficient suction should be immediately available for removal of clots or tumor fragments. These patients also have an increased risk of perioperative lung infections due to aspiration of blood or tumor. Surgery may involve tracheal resection and airway management should be planned accordingly.

Q. How will you assess for retrosternal extension of thyroid mass?

Symptoms:

- Dyspnea, choking, hoarseness, dysphagia
- Unable to "get below the swelling"
- Percussion over the manubrium sternum (seldom followed in current practice)
- Lateral neck X-ray
- CT scan.

SVC obstruction: The patient will have edema and dilated veins on the head, neck, and upper part of the chest. Severe cases may have respiratory distress due to airway edema. Increased intraoperative bleeding is expected.

Large-bore venous access should be secured in lower limbs or femoral vein.

Pemberton's sign is the development of facial flushing, distended neck and head superficial veins, inspiratory stridor and elevation of the jugular venous pressure (JVP) upon raising of the patient's both arms above his/her head simultaneously, as high as possible. A positive sign is indicative of SVC syndrome. In current practice, with the widespread use of CT scan, this test is rarely performed as it can be dangerous in the OPD setting.

Q. Which investigations will you prescribe and why? What is the role of flow volume loops?

Ans.

Hemoglobin: Baseline. To estimate the maximum allowable blood loss and to detect anemia as antithyroid medication may cause bone marrow suppression.

Thyroid function tests: To confirm the euthyroid state (to detect subclinical abnormality). Patients with hypothyroidism on stable thyroxine dose with documented

normal TFTs in the preceding 6 months do not require repeat TFT testing prior to surgery, if clinically euthyroid. Patients with Graves' disease in remission should have TFTs as relapses are known:

- ECG: Screening for IHD/dysrhythmias
- Serum creatinine if patient > 60 years
- X-ray chest.

CT neck: The anesthetist should always review the anatomy on CT scan images to assess airway status.

X-ray neck AP/lateral if CT neck not available—for tracheal deviation/compression.

Flow volume loop may detect subclinical airway obstruction and also suggest the type and site of obstruction, i.e., fixed/variable, intrathoracic/extrathoracic. In clinical practice, with the availability of CT scan images, flow volume loops do not contribute much to the management in suspected airway compromise.

Indirect laryngoscopy is used to diagnose the preexisting vocal cord palsy.

Q. What are the principles of anesthetic management for this patient?

Ans.

- Airway management at the induction of anesthesia, maintenance, and during the recovery period
- Management of complications of hypo-/hyperthyroidism
- Management of problems due to retrosternal extension.

Q. What monitoring will you institute?

Ans.

- ECG, SpO₂, ETCO₂, core temperature, BP.
- Invasive arterial pressure monitoring if the patient is hyperthyroid or major blood loss is expected.

Q. How will you manage the airway in this patient?

Ans. Preparation: Preparation and planning are of the utmost importance when dealing with a difficult airway. Alternate plans of action, if the primary plan fails, should be discussed by the anesthesia and surgical team to prevent panic and delays in management.

The patient might have airway obstruction due to external pressure, invasion into the trachea, severe deviation, or vocal cord palsy.

Problems anticipated

Deviation: Distorted anatomy—visualization of the larynx may be difficult.

Compression of the trachea: The patient may be unable to lie supine. A small-sized endotracheal tube (ETT) may be required.

In a large thyroid, emergency airway by cricothyroid puncture or tracheostomy may be difficult or impossible. However, in very severe obstruction, tracheostomy under

local anesthesia should be considered by the surgeon after careful review of the distorted anatomy. Ultrasound may be useful in identifying the trachea and the vascular structures near the trachea.

Review CT scan—to assess the site and extent of tracheal/bronchial obstruction and plan airway management.

A preoperative awake fiberoptic bronchoscopy may help to assess the airway involvement and help decide the induction and intubation technique. If a flexible/fiberoptic bronchoscope (FOB) is not available, some anesthesiologists perform laryngoscopy under topical anesthesia to assess the anatomy of the larynx and decide the plan for securing the airway and management of anesthesia:

- Avoid sedative premedication.
- Difficult airway kit should be ready.
- Keep small size flexometallic tubes handy.
- A rigid bronchoscope (and a skilled operator) should be immediately available if required to hold the airway open and allow ventilation if airway collapse occurs on the induction of anesthesia. Turning the patient lateral or using forceps to lift thyroid away may also help to relieve the compression.
- Equipment for cricothyroidotomy/tracheostomy. Its role in emergency airway control in large anterior neck mass is limited.
- An experienced surgeon should be immediately available.

In case of a compromised airway, an inhalational induction with sevoflurane in oxygen is the preferred technique as it preserves spontaneous breathing. IV agents are generally avoided as they are more likely to cause apnea. Muscle relaxants should be used very judiciously when airway difficulties are anticipated. In patients who cannot lie supine due to airway obstruction, intubation may have to be done in a semisitting position and induction of anesthesia in this position can lead to severe hypotension.

In severe displacement of the trachea, a fiberoptic-guided intubation under topical anesthesia is the recommended technique. However, this technique should be used with caution in severe obstruction as the small caliber of the airways may sometimes make the passage of adult fiberoptic scope difficult and induce complete obstruction.

Controlled tracheostomy following a femorofemoral cardiopulmonary (CP) bypass that had been initiated under local anesthetic has been described in a case of airway obstruction with supraglottic edema due to a large thyroid mass. This is described as the ultimate solution to the problem of a difficult airway. However, it should be understood that a "stand by" CP bypass may not be able to rescue a "cannot intubate," "cannot oxygenate" situation. When indicated, the femoral vessels should be cannulated and CP bypass circuit primed and ready for use.

A standard polyvinylchloride (PVC)-cuffed ETT is adequate in most cases. A flexometallic tube is preferred when there is tracheal compression and if possible the ETT should be placed beyond the length of (lower limit of) compression. It may also be useful in a tortuous airway. Extra care should be taken to secure the flexometallic tube as it is more prone to dislodgement.

Opioid with a shorter duration of action, e.g., fentanyl, is preferred in order to have minimal sedative effects at recovery. Isoflurane and sevoflurane are volatile agents of choice. Muscle relaxants (e.g., vecuronium) are guided by peripheral nerve stimulators.

If intraoperative RLN monitoring is practiced, muscle relaxants are avoided.

Other perioperative considerations:

- The eyes should be protected, especially if exophthalmos is present.
- The patient is positioned headup to help venous drainage.
- The neck is hyperextended and should be well supported to avoid strain on cervical spine ligaments.
- Extension tubing for IV lines and long respiratory hoses may be required.
- Valsalva maneuver in Trendelenburg position is carried out to check hemostasis.
- Steroids may be given if extensive tracheal handling and edema are suspected.

Extubation should be smooth and coughing should be avoided to prevent bleeding. The surgeon may wish to observe the movement of the vocal cords at the end of the surgery. Though widely practiced in the past, it is not much favored in current practice. This could be due to the difficulty in performing an adequate laryngoscopy in a patient with recovering neuromuscular function and light plane of anesthesia and the attendant sympathetic stimulation. Also, vocal cord status at the end of surgery has been shown to be a poor predictor of postoperative cord function. If the assessment of vocal cord function is deemed necessary, flexible laryngoscopy may be more useful. Ultrasound imaging has also been used to observe the vocal cord movement.

At extubation, the possibility of tracheomalacia (due to the weakness of the tracheal rings) and vocal cord palsy should be kept in mind and the patient should be observed closely. Tracheomalacia should be suspected in long-standing goiters and the surgeon may palpate the trachea intraoperatively to assess the quality of tracheal cartilages. Tracheomalacia is defined as dynamic airway collapse with a decrease in the tracheal lumen by >50%. It causes collapse of the extrathoracic trachea during inspiration and collapse of the intrathoracic trachea in expiration. Extubation should be performed after adequate preoxygenation and with

preparation for reintubation if required. An ETT may be slowly withdrawn and signs of obstruction watched for. In difficult intubation, it is advisable to extubate over an airway exchange catheter. Flexible laryngoscopy during spontaneous respiration in an extubated trachea or through a laryngeal mask airway (LMA) is useful for the diagnosis of respiratory obstruction due to tracheomalacia or vocal cord palsy.

Q. How is the leak test performed?

With the ETT in place, the cuff is deflated. A leak around the ETT can be detected on intermittent positive pressure ventilation (IPPV) by palpating over the trachea or observing for the difference between the set and expired tidal volume. During spontaneous respiration, the patient should be able to breathe around the tube when the tubal lumen is plugged, suggesting free space around the tube.

The leak test has been described for airway edema and its usefulness in tracheomalacia is controversial.

Q. What is the management of tracheomalacia?

Ans. Post-thyroidectomy, tracheomalacia is rare in developed countries but has a higher incidence in countries such as India where patients do present with large goiters.

Tracheomalacia causing airway obstruction requires intubation. Noninvasive continuous positive airway pressure (CPAP) may help to relieve symptoms in mild cases. Fibrosis around the trachea stabilizes the tracheal wall but may take several days. Tracheostomy may be required in some cases. Tracheal stents or surgical repair may be required in patients with a persistent problem.

Q. What is the postoperative analgesia plan?

Ans. Severe pain is uncommon and simple analgesics such as paracetamol and nonsteroidal anti-inflammatory drugs (NSAIDs) are usually sufficient. Wound infiltration with bupivacaine may also be used.

Q. What is the anesthesia management for intraoperative nerve monitoring (IONM) in thyroid surgery?

Ans. In IONM in thyroid surgery, the RLN is stimulated and electromyographic (EMG) signals are picked up from the vocal cords which help in the identification of RLN, thereby avoiding injury. IONM is increasingly being used during thyroid surgery, particularly in patients with malignant tumors. However, a recent Cochrane database review has shown no conclusive evidence for the superiority or inferiority of IONM over visual nerve identification for RLN injury. The two main components of IONM are nerve stimulation and assessment of vocal fold response to nerve stimulation. The stimulation of RLN can be done with:

- Intermittent stimulation—a low-voltage electric current delivered by a handheld probe
- Continuous stimulation—an electrode attached to the ipsilateral vagus nerve.

The vocal fold response to nerve stimulation is measured by a laryngeal surface electrode system either built into or attached to the ETT (**Fig. 1**).

During surgery, the vocal fold response to nerve stimulation is displayed on a nerve monitor as auditory or visual EMG signals. Change in the EMG signal pattern alerts the surgeon to possible RLN irritation. Before starting the IONM, primary vagal stimulation is done to confirm correct ETT placement and to rule out any preexisting defects in vocal cord activity and other confounding factors such as residual neuromuscular junction blockade (NMB) and device malfunction.

Anesthesia concerns during thyroid surgery under IONM are:

- NMB interferes with monitoring as paralyzing agents will interfere with obtaining of an EMG signal. Therefore, *neuromuscular conduction should be normal at the time of nerve monitoring*. The strategies for NMB management for IONM are as follows:
 - Avoiding NMB completely. Intubation is performed without a muscle relaxant. This technique needs expert care to avoid violent coughing and pressor response.
 - Using succinylcholine to facilitate endotracheal intubation, thus allowing rapid termination of NMB. However, one has to consider the side effects and contraindications of succinylcholine.
 - Using a single induction dose of short-/intermediate-acting nondepolarizing neuromuscular blocking agents (NMBAs) for intubation and allowing spontaneous recovery. This is ideal if the time
- interval between intubation and IONM is around 45–60 minutes. A peripheral nerve stimulator is used to confirm complete recovery of the neuromuscular junction.
- Using rocuronium for intubation. The reversal of NMB is achieved with sugammadex at any point in time after rocuronium administration. This strategy has the dual advantage of good intubating conditions and complete neuromuscular junction recovery immediately, without having to wait for the elimination of the muscle relaxant. In India, the availability and high cost of sugammadex are prohibiting factors.
- Anesthetic agents.
 - Inhalational anesthetic agents do not interfere with triggered EMG signals and can be used during thyroid surgeries (unlike neurosurgery IONM cases).
 - TIVA with propofol and remifentanyl is advised for neurosurgery IONM and can be used in thyroid surgeries also. If remifentanyl is not available, TIVA using propofol, fentanyl, and small doses of dexmedetomidine provides good conditions for IONM.
- It is advisable (but not mandatory) to use a videolaryngoscope for intubation to confirm the accurate positioning of electrodes assembly. The ETT should be positioned such that the electrodes embedded in the ETT should come in contact with vocal cords. Hyperextension of the neck for surgical exposure may cause outward movement of the tube, and therefore a check laryngoscopy is advisable to confirm the correct ETT position.

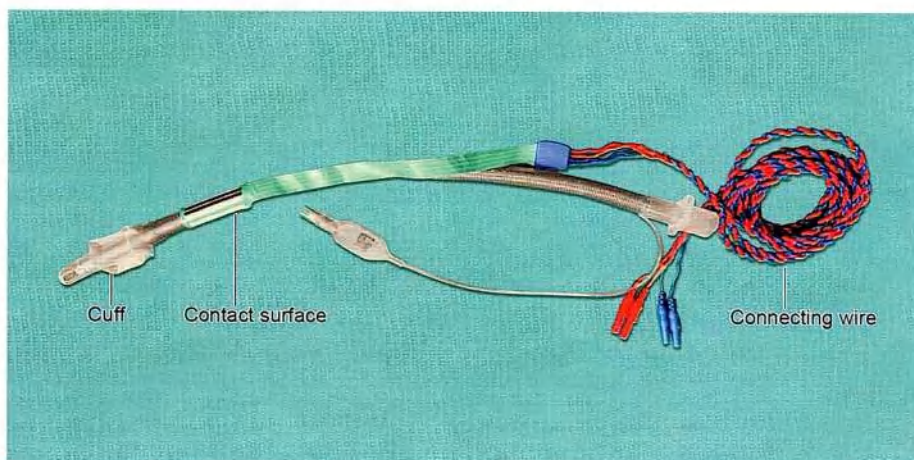


Fig. 1: Electromyography endotracheal tube.

Q. What are the problems of positioning for thyroid surgery?

Ans. Thyroid surgery requires hyperextension of head. Care should be taken to support the head adequately to prevent strain on cervical spine ligaments and spine injury. In patients with restricted or painful neck extension or severe osteoporosis, the limit of comfortable neck extension should be assessed prior to anesthesia and extreme position is avoided during intubation and surgery.

Arms should be adducted by the patient's sides whenever possible. When arms are positioned at right angles, care should be taken to prevent hyperextension at the shoulder joint which can stretch the brachial plexus.

Q. What are the possible intraoperative problems? Discuss their management.

Ans.

- *Increased airway resistance (causes):*
 - Kinking of the tube, secretions obstructing the tube, endobronchial intubation, bronchospasm
 - Surgeons manipulating the trachea during dissection
 - External compression of trachea/bronchi distal to the ETT in cases of large retrosternal thyroid
 - Rarely, a part of the intratracheal tumor gets dislodged during intubation or manipulation by the surgeon and migrates distally into the tracheal or bronchial lumen causing an obstruction.
- *Accidental extubation/disconnection:*
 - Hyperextension of the head causes ETT to migrate outward.
 - Surgeons operating near the airway, so more chances of airway accidents.
- *Thyroid crisis.*

Q. Can thyroid surgery be done as day surgery?

Ans. It is not routinely recommended as postoperative airway problems are possible. It may be considered in a select group of patients and where a very good community medical support system is available.

Q. What are the possible postoperative problems and their management?

Ans.

Respiratory Distress

Causes:

- Specific to thyroid surgery:
 - Airway obstruction:
 - *Wound hematoma:* Traditionally, suture cutters were kept at the bedside to enable rapid relief of a hematoma. It is essential to open all layers to relieve the obstruction. Early intubation is advisable when significant hematoma is present

as airway compression and edema (hematoma obstructs venous drainage causing edema) may worsen rapidly

- RLN damage
- *Tracheomalacia causing dynamic airway obstruction:* Urgent reintubation, possibly tracheostomy, and some form of tracheal support
- *Laryngeal edema (uncommon):* Mostly related to large hematoma obstructing venous drainage. Other causes are trauma during intubation and myxedema
- *Hypocalcemia causing laryngospasm:* Hypocalcemia usually occurs at about 36 hours post-operatively, usually temporary, unless there is severe damage to all the parathyroid glands. Treatment includes 10 mL of 10% calcium gluconate. Long-term calcium supplementation is required.

- All causes of postanesthesia respiratory distress.

Metabolic Disturbances

- *Hypothyroidism:* May need thyroxine supplements.
- *Hypoparathyroidism:* This may be due to direct trauma to the parathyroid glands, devascularization of the glands, or removal of the glands during surgery.
- *Hypocalcemia:* The cause of transient hypocalcemia after surgery is not clearly understood. It may be attributable to temporary hypoparathyroidism caused by reversible ischemia to the parathyroid glands. Other hypotheses to account for transient hypocalcemia not caused by hypoparathyroidism include calcitonin release and hungry-bone syndrome.

Q. Describe the nerve supply of larynx.

Ans.

- Most of the muscles of the larynx receive their innervation via the RLN branch of the vagus nerve except the cricothyroid muscle which receives its innervation via the external laryngeal nerve. Cricothyroid muscle is important for tensing of the vocal cord.
- Sensory innervation of the larynx is via branches of the vagus nerve. Above the vocal folds, the sensory innervation of the larynx is via the internal laryngeal nerve. Below the vocal folds, it is by the branches of the RLN.
- Parasympathetic innervation is mainly by the branches of the vagus nerve.

Q. Enumerate causes and describe common nerve injuries after thyroid surgery.

Ans. Causes: Contusion, traction, entrapment, actual transection, and ischemia.

External Branch of the Superior Laryngeal Nerve

Injury causes a change of voice quality and loss of vocal stamina. Treatment involves speech therapy.

Recurrent Laryngeal Nerve

Unilateral injury: Unilateral cord palsy causes hoarseness and a weak cough but usually without significant airway obstruction at rest. The patient has a risk of aspiration. Compensatory hyperadduction by the opposite cord may allow adequate vocal and sphincter function.

Bilateral injury: Bilateral injury of the abductors can cause unopposed adduction of cords causing complete glottic closure. The patient will need immediate tracheostomy. In bilateral complete denervation, both vocal cords are in cadaveric position. Phonation is severely affected. Stridor (predominantly inspiratory) may be present and the patient carries a very high risk of aspiration:

- Initial treatment involves ensuring an adequate airway. Endotracheal intubation is the first line of management. IV steroids may be helpful in some cases of reversible damage.
- A trial of extubation may be performed after several days in a controlled setting as reintubation may be necessary. Extubation over an airway exchange catheter is advisable if the patient has a difficult upper airway. If nerve function has not recovered after the second trial of extubation, tracheostomy is warranted.
- Corrective procedures are generally not performed for at least 6 months after surgery as delayed recovery is possible.
- Treatments of vocal cord paralysis include cordotomy (relieves stridor but increases aspiration risk), intracordal injection, laryngeal framework surgery, thyroplasty, and laryngeal reinnervation.

Q. What is the role of cervical plexus blocks and cervical epidural anesthesia in thyroid surgery?

Ans. Cervical plexus blocks and cervical epidural anesthesia in thyroid surgery are controversial. Regional techniques may be possibly used in cooperative patients with small nonmalignant thyroid tumors.

Proponents cite avoiding airway manipulations and monitoring vocal cord function as a major advantage of the technique. Unilateral combined superficial and deep cervical plexus blocks have been performed for unilateral surgery. A bilateral combined block is contraindicated because of the risk of bilateral phrenic nerve palsy. Superficial cervical plexus blocks have been tried unilaterally and bilaterally for analgesia, but the reports are conflicting regarding their efficacy. Superficial cervical plexus blocks have been used for video-assisted thyroidectomy.

Cervical epidural catheters have been used in some reports. However, the risk of bradycardia, hypotension, and acute ventilatory failure related to respiratory muscle paralysis is high and the patient needs close monitoring.

Q. What are the implications of pregnancy in hyperthyroidism?

Ans. During pregnancy, the increase in TBG causes an increase in total serum thyroid hormones which can lead to an erroneous diagnosis, so free T_4 and T_3 levels should be used to determine thyroid status.

Both PTU and methimazole cross the placenta and can affect fetal thyroid function, especially at higher doses. PTU is less teratogenic than methimazole. Most women with Graves' disease can be treated medically during pregnancy, with a target T_4 level at or slightly higher than the upper normal limit (and the lowest effective dose) to ensure normal thyroid hormone levels in the fetus. Iodides cause fetal hypothyroidism and are contraindicated, so is radioactive iodine.

Graves' disease generally improves in the second and third trimesters of pregnancy, allowing reduction or discontinuation of antithyroid drug therapy, although it can exacerbate in the postpartum period.

During pregnancy, ultrasonography (USG) monitoring is used to check for the presence of fetal goiter. Fetal goiter can indicate excessive antithyroid drug treatment in the mother or fetal Graves' disease. Maternal complications of Graves' disease in pregnancy include preeclampsia and preterm delivery.

Women receiving thyroxine therapy for hypothyroidism should have their dose increased by up to 50% during pregnancy. Avoiding maternal (and fetal) hypothyroidism is extremely important because of potential damage to fetal neural development, an increased incidence of miscarriage, and preterm delivery.

SUGGESTED READING

1. Arora N. Seminars: Local and regional anesthesia for thyroid surgery. *J. Surg Oncol.* 2006;94:708-13.
2. Bacuzzi A. Anaesthesia for thyroid surgery: perioperative management. *Int J Surg.* 2008;6:S82-S5.
3. Cooper D. Antithyroid drugs. *N Engl J Med.* 2005;352:905-17.
4. Farling PA. Thyroid disease. *Br J Anaesth.* 2000;85:15-28.
5. Kahalya G, Bartalenab L, Hegedüs L, Leenhardt L, Poppe K, Pearce SH. 2018 European Thyroid Association Guideline for the management of Graves' hyperthyroidism. *Eur Thyroid J.* 2018;7:167-86.
6. Mayilvaganan S, Agarwal A. Management of post-thyroidectomy tracheomalacia. *WJOES.* 2014;6:96-8.
7. The Association for Clinical Biochemistry and British Thyroid Association. (2006). UK Guidelines for the Use of Thyroid Function Tests, 2006. [online] Available from https://www.british-thyroid-association.org/sandbox/bta2016/uk_guidelines_for_the_use_of_thyroid_function_tests.pdf. [Last accessed February, 2020]

Diabetes Mellitus

Madhavi Shetmahajan, Sohan Lal Solanki

A 30-year-old gentleman, known case of diabetes mellitus (DM) since last 15 years on insulin, presented to outpatient department with abdominal pain. CT scan confirmed a stomach tumor and is posted for total gastrectomy.

A 70-year-old gentleman, a known case of DM since 25 years, currently on oral hypoglycemic drugs is posted for emergency incision and drainage of a large thigh abscess.

Q. How is diabetes classified?

Ans. American Diabetes Association (ADA), 2014, classification of diabetes mellitus (DM) is as follows:

- Type 1 diabetes (β -cell destruction, usually leading to absolute insulin deficiency):
 - Immune-mediated
 - Idiopathic
- Type 2 diabetes (could be predominantly insulin resistance with relative insulin deficiency to a predominantly secretory defect with insulin resistance)
- *Other specific types:*
 - Genetic defects of β -cell function
 - Genetic defects in insulin action
 - Diseases of the exocrine pancreas
 - Endocrinopathies
 - Drug or chemical-induced
 - Infections
 - Uncommon forms of immune-mediated diabetes
 - Other genetic syndromes sometimes associated with diabetes
- Gestational diabetes mellitus (GDM).

Immune-mediated Type 1 Diabetes

Terms used previously include insulin-dependent diabetes or juvenile-onset diabetes. This results from a cellular-mediated autoimmune destruction of the β -cells of the pancreas. Commonly presents in childhood and adolescence. The rates of β -cell destruction are variable, rapid—mainly in children, and slow—mainly in adults. Those with complete destruction of β -cell are dependent on insulin supplementation for survival. Ketoacidosis is a common presentation. These patients are prone to other

autoimmune diseases such as Graves' disease, Hashimoto's thyroiditis, Addison's disease, autoimmune hepatitis, myasthenia gravis, and pernicious anemia.

Idiopathic Type 1 Diabetes

Idiopathic type 1 diabetes have insulinopenia but no evidence of autoimmune activity.

Type 2 Diabetes

Type 2 diabetes includes ~90–95% of those with diabetes. Terms used previously include noninsulin-dependent diabetes and adult- or maturity-onset diabetes. These patients have insulin resistance with inadequate insulin secretion to compensate for the increased requirement (relative insulin deficiency). Most do not need insulin treatment for survival. Advancing age, obesity, sedentary lifestyle, hypertension, previous gestational diabetes, and genetic makeup are predisposing factors. Ketoacidosis is unusual unless precipitated by stress such as infection.

Other Specific Types

Endocrinopathies

The abnormal secretion of certain hormones antagonizes insulin action, for example, acromegaly, Cushing's syndrome, glucagonoma, pheochromocytoma, hyperthyroidism, somatostatinoma, and aldosteronoma.

Drug-induced Diabetes Mellitus

Acts by impairment of insulin action or reduction in insulin secretion, for example, β -adrenergic agonists, thiazides, glucocorticoids, thyroid hormone, diazoxide, and phenytoin. α -interferon acts by developing islet cell antibodies.

Exocrine Pancreatic Diseases

Exocrine pancreatic diseases are pancreatitis, trauma, infection, pancreatectomy, and pancreatic carcinoma.

Q. What is metabolic syndrome?

Ans. "Metabolic syndrome" (also called syndrome X or insulin resistance syndrome): It is a noncausally linked cluster of symptoms which carries a high-risk of macrovascular disease. The cluster includes impaired glucose tolerance or diabetes, hypertension, dyslipidemia, and abdominal obesity.

Q. Why is it important to know the type of diabetes?

Ans. The risk of complications such as ketoacidosis and the perioperative management of blood sugar may differ in type 1 and type 2 diabetes. However, the quality of glycemic control is likely to be more important than the etiology for development of complications. The type 1 patients require basal insulin (and glucose) supplementation to prevent catabolism. They usually have less insulin resistance and tend to require lower insulin doses for same glucose load than insulin-treated type 2 diabetics. However, type 1 patients tend to have more labile blood sugar concentrations during periods of stress than type 2 diabetics.

In secondary diabetes, the various endocrine abnormalities may have perioperative implications.

Q. What other details should you seek in the clinical evaluation of a diabetic patient?

Ans. Duration of diabetes, medication history and quality of glycemic control, episodes of hypoglycemic or hyperglycemic complications, and signs and symptoms of organ dysfunction associated with diabetes.

Complications of diabetes depend on the blood sugar control and duration of diabetes. Good blood sugar control is known to reduce risk of microvascular complications though the evidence for reduced risk of macrovascular complications is less robust.

Q. What are the complications of diabetes?

Ans. Hyperglycemia is believed to cause its complications through formation of abnormal glycosylation end products, formation of large molecules causing intracellular swelling, and causing vasodilatation which obtunds autoregulation.

The important complications for the anesthetist are coronary heart disease, diabetic nephropathy, and autonomic neuropathy because these may have a direct effect on the development of perioperative complications. The complications can be grouped as:

- *Macrovascular (coronary artery disease, peripheral arterial disease, and stroke):*

- Mechanisms of injury:
 - Atherosclerosis
 - Increased coagulability and impaired fibrinolysis
- *Microvascular complications (diabetic nephropathy, neuropathy, and retinopathy):*
 - Mechanisms of injury:
 - Osmotic stress from sorbitol accumulation
 - Formation of advanced glycosylated end products
 - Free radical production and reactive oxygen species formation
- *Diabetic nephropathy:*
 - Mechanism of injury:
 - Glomerular hyperfiltration
 - Increase in glomerular hydrostatic pressure causes glomerular damage and microalbuminuria
 - Impaired endothelium-dependent vasodilatation
 - The earliest sign is microalbuminuria.

Angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers have protective action.

- *Diabetic neuropathy:* Chronic sensorimotor distal symmetric polyneuropathy—most common symptoms are burning, tingling, and numbness. Mononeuropathies are sudden in onset. Median, ulnar, and radial nerves are commonly affected.
- *Diabetic amyotrophy:* Severe pain and muscle weakness and atrophy, usually in thigh muscles.
- *Diabetic autonomic neuropathy (DAN):* Autonomic dysfunction is detectable in up to 40% of type 1 and 17% of type 2 diabetic patients. Mechanisms suggested include local ischemia, tissue accumulation of sorbitol, altered function of neuronal Na^+/K^+ -ATPase pump activity, and immunologically mediated damage.
- *Diabetic retinopathy:*
 - Dot hemorrhages, hard exudates, microaneurysms, and retinal edema
 - Proliferative retinopathy and retinal detachment.

Cardiac Complications

The diabetic patient has an increased risk of:

- *Coronary artery disease:*
 - Diabetic patients >65 years of age have significant symptomatic/asymptomatic coronary artery disease. Silent ischemia may be present because of DAN.
- Hypertension
- Peripheral arterial disease

- *Systolic and diastolic dysfunction, related to:*
 - Coronary artery disease
 - Hypertension
 - Left ventricular hypertrophy
 - Endothelial dysfunction
 - Obesity
 - Autonomic neuropathy.

Central Nervous System

- *Cerebral vascular disease related to:*
 - Hypertension
 - Dyslipidemia
 - Accelerated atherosclerosis
 - Abnormal endothelial proliferation
 - Increased coagulability and impaired fibrinolysis.

Renal dysfunction: It is related to hypertension, dyslipidemia, and anemia. Iatrogenic exposure to nephrotoxins and cardiac surgery are responsible for precipitating renal failure.

Contrast-induced nephropathy (CIN): Avoiding dehydration and nephrotoxins, and rational use of contrast-based radiographic procedures may help to prevent worsening of renal damage. Volume expansion with isotonic solution, e.g., normal saline has been shown to be beneficial. Current data is not sufficient to support the use of hemofiltration, renal-dose dopamine, osmotic diuresis, or calcium channel blockade for renal protection. A recent meta-analysis found that N-acetyl cysteine (NAC) or a NAC statin combination when added to intravenous (IV) saline reduced the risk of CIN.

Gastrointestinal System

Risk of aspiration is increased because of autonomic neuropathy (decreased ability to coordinate swallowing and/or gastroparesis).

Respiratory System

Early investigations show that diabetes affects the respiratory system but evidence regarding its impact on functional status of the patient is lacking. The underlying mechanisms hypothesized include impaired lung elasticity, pulmonary microangiopathy, and chronic systemic inflammation of diabetes. Some of the reported lung dysfunctions attributed to diabetes are:

- Decreased lung volume and lung diffusing capacity
- Reduced hypoxic-induced ventilatory drive (in patients with autonomic neuropathy)
- Diabetics may be prone to respiratory depression from opioids and sedative agents due to impaired chemoreceptor activity
- Airway difficulties.

Q. What airway problems are possible and why? Which specific tests can be done to predict airway difficulties?

Ans. The “stiff joint” or “limited joint mobility” (LJM) syndrome or diabetic cheiroarthropathy:

It manifests with joint rigidity (joints supporting the airway such as the temporomandibular, atlanto-occipital, and cervical spine joints are most commonly involved). Neck extension and laryngoscopy may be difficult.

Cause: Nonenzymatic glycosylation of proteins and abnormal cross-linking of collagen in joints and other tissues.

About one-third of patients with long-term type 1 diabetes are reported to undergo a difficult laryngoscopy and intubation.

Tests

“Prayer” sign: Ability to oppose the palms and fingers as in prayer. Diabetic patients with stiff joints have a positive test as they are unable to do this.

Palm print test: Stiffness of the fourth and fifth interphalangeal joints causes alteration in palm print:

- The palm and fingers of the dominant hand of the patient is painted with ink. Then, the hand is firmly pressed on a white sheet of paper on a hard surface.
- **Scoring:**
 - Grade 0—all phalangeal areas visible
 - Grade 1—deficiency in the interphalangeal areas of 4th and/or 5th digit
 - Grade 2—deficiency in the interphalangeal areas of 2nd to 5th digit
 - Grade 3—only the tips of digits seen.

Q. How will you assess for autonomic neuropathy?

Ans. Important clinical signs are resting tachycardia, lack of sweating, early satiety, sexual dysfunction, and lack of heart rate variability (HRV).

Signs and symptoms of autonomic disease can be classified as given below:

Cardiovascular

- Resting tachycardia
- Exercise intolerance
- Orthostatic hypotension
- Silent myocardial ischemia.

Gastrointestinal

- Esophageal dysmotility (dysphagia)
- Gastroparesis
- Constipation
- Diarrhea
- Fecal incontinence.

Genitourinary

- Neurogenic bladder (diabetic cystopathy)
- Erectile dysfunction
- Retrograde ejaculation
- Female sexual dysfunction (e.g., loss of vaginal lubrication).

Metabolic

- Hypoglycemia unawareness
- Hypoglycemia-associated autonomic failure.

Sudomotor

- Anhidrosis
- Heat intolerance
- Gustatory sweating
- Dry skin.

Pupillary

- Pupillomotor function impairment (e.g., decreased diameter of dark adapted pupil)
- Argyll-Robertson pupil.

The differential diagnosis of DAN:

- Pure autonomic failure (idiopathic orthostatic hypotension)
- Multiple system atrophy with autonomic failure (Shy-Drager syndrome)
- Addison's disease and hypopituitarism
- Pheochromocytoma
- Hypovolemia
- Medications with anticholinergic or sympatholytic effects (vasodilators and sympathetic blockers)
- Peripheral autonomic neuropathies (e.g., amyloid neuropathy, idiopathic autonomic neuropathy).

Q. Describe tests for DAN.*Ans.*

- *Early stage:* Abnormality of heart rate response during deep breathing alone
- *Intermediate stage:* An abnormality of Valsalva response
- *Late stage:* The presence of postural hypotension
- The tests are valid as markers of autonomic neuropathy, if following factors ruled out:
 - End-organ failure
 - Concomitant illness
 - Drug such as antidepressants, antihistamines, diuretics, vasodilators, sympathetic blockers, and vagolytics.

Tests for Parasympathetic Control

Heart rate variability in response to:

- Deep breathing
- Standing
- Valsalva maneuver.

Heart rate variability methods are age-dependent but independent of the intrinsic heart rate. They are the standard screening methods for autonomic dysfunction. Valsalva maneuver is influenced by both parasympathetic and sympathetic activity.

Tests for Sympathetic Control

Blood pressure response to:

- Standing or passive tilting
- Sustained handgrip
- *Response to the Valsalva maneuver:* In the standard Valsalva maneuver, the supine patient, connected to an ECG (electrocardiogram) monitor, forcibly exhales for 15 seconds against a fixed resistance (40 mm Hg) with an open glottis. This results in a sudden transient increase in intrathoracic and intra-abdominal pressures, with a characteristic hemodynamic response.
 - *Phase I:* Transient rise in blood pressure and a fall in heart rate due to compression of the aorta and propulsion of blood into the peripheral circulation. Hemodynamic changes are due to mechanical factors.
 - *Phase II:* Initial fall in blood pressure followed by the recovery of blood pressure later in the phase. The blood pressure changes are accompanied by an increase in the heart rate. There is a fall in cardiac output due to decreased venous return which causes reflex tachycardia and increased peripheral resistance.
 - *Phase III:* Drop in blood pressure and rise in heart rate when expiration stopped.
 - *Phase IV:* Overshoot of blood pressure above the baseline value due to residual vasoconstriction in a setting of a now normal venous return and cardiac output. There is a reflex bradycardia.

The Valsalva ratio is calculated from the ECG waveform by dividing the longest R-R interval after the maneuver (in phase IV) to the shortest R-R interval during the maneuver. A Valsalva ratio < 1.2 is abnormal.

Heart Rate Variability

- Respiratory sinus arrhythmia (RSA) is a normal phenomenon due to vagal input to sinus node during expiration causing cardio deceleration. Autonomic neuropathy decreases the HRV (difference in between maximum and minimum heart rate during the respiratory cycle). Specialized monitors are available to accurately determine the HRV. A clinical but less accurate method is to ask the patient to breathe quietly and deeply at a rate of 6 breaths/min (at this rate there is maximum variation in heart rate):

- Normal variability >15 beats/min
- Abnormal result <10 beats/min.

Aging is associated with decreased RSA due to decreased vagal tone and decreased beta receptor responsiveness and the criteria may not apply to patients above 60 years of age.

- **Response to standing up (30:15 ratio):** There is a rapid increase in heart rate in response to standing that is maximal at approximately the 15th beat after standing. This is followed by a relative bradycardia that is maximal at approximately the 30th beat after standing. The heart rate should increase by 10%. 30:15 ratio is measured as the longest R-R interval during beats 20–40 divided by the shortest R-R interval during beats 5–25. In patients with DAN, there is only a gradual increase in heart rate:
 - 30:15 ratio >1.04 is normal.
 - 1.01–1.03 is borderline.
 - <1.01 is abnormal.

Orthostatic hypotension: It is defined as a fall in blood pressure > 30 mm Hg for systolic blood pressure (SBP) or > 10 mm Hg for diastolic blood pressure in response to postural change, from supine to standing. Normally, blood pressure is rapidly corrected by baroreflex-mediated peripheral vasoconstriction and tachycardia and the fall in SBP is <10 mm Hg. A drop by 11–29 mm Hg is borderline and a drop >30 mm Hg is significant.

Response to tilting: It is a more precise stimulus and may be used instead of standing. The response is similar.

Sustained handgrip: Sustained muscle contraction causes a rise in systolic and diastolic blood pressure and heart rate. Exercising muscle stimulates a reflex arc resulting in increased cardiac output and heart rate. As the peripheral vascular resistance is maintained the diastolic pressure rises by >16 mm Hg. A response of <10 mm Hg is considered abnormal.

Gastroparesis: The finding of retained food in the stomach after an 8–12 hour fast in the absence of obstruction is diagnostic of gastroparesis.

Management of DAN: Control of blood sugar levels, ACE inhibitors, and β -blockers have been tried for control of the condition.

Q. How does autonomic neuropathy affect the perioperative course?

Ans. Autonomic neuropathy affects compensatory mechanisms of the cardiovascular system (e.g., baroreceptor reflexes) increasing risk of hemodynamic lability, particularly with change in patient position, initiation of positive pressure ventilation, and institution

of sympathetic blockade as in neuraxial blocks. Close monitoring is required along with meticulous maintenance of intravascular volume. Loss of HRV may be a contributory risk factor for ventricular arrhythmias and sudden death in these patients. Patients with autonomic neuropathy are likely to have silent myocardial ischemia. Respiratory arrest is also seen in patients with autonomic neuropathy.

Q. What do you want to know about the patients' antidiabetic treatment?

Ans.

- **Preoperative control of blood sugar:** The preoperative control of blood sugar is best assessed with HbA1c. The Association of Anaesthetists of Great Britain and Ireland 2015 guidelines on perioperative management of the surgical patient with diabetes advocate HbA1c < 69 mmol/mol (<8.5%) for elective surgery.
- Details of oral hypoglycemic agents.
- If insulin, type of insulin and dosing schedule. The groups of oral hypoglycemic drugs and their perioperative implications are:
 - **First-generation agents:** For example, tolbutamide, tolazamide, and chlorpropamide. Have long duration of action.
 - **Second-generation agents:** For example, glipizide, glyburide, glimepiride, gliclazide, and glibenclamide. Glibenclamide has a longer duration of action and more likely to cause hypoglycemia especially in presence of renal insufficiency.
 - The potassium channel-blocking effect of sulfonylureas may interfere with myocardial ischemic preconditioning thereby increasing the risk of cardiac complication especially in major surgery and critical illness.
- **Meglitinides:** Stimulate insulin secretion partly in a similar way to sulfonylureas but are shorter-acting and have a more rapid onset of action, e.g., repaglinide.
- **Biguanides:** Metformin is the first-line treatment in type 2 DM. Its mechanism of action is sensitizing the liver to insulin and reducing liver glucose output. It also improves insulin sensitivity in peripheral tissues and decreases serum free fatty acid concentrations. Metformin is well tolerated and is less likely to cause hypoglycemia than sulfonylureas or insulin.

Lactic acidosis associated with metformin is rare but has a high mortality rate. Therefore, metformin should be prescribed with caution in patients with moderate to severe renal or liver dysfunction, alcohol abuse, poor cardiac function, and those with compromised tissue perfusion.

The Society of Ambulatory Anesthesia consensus guidelines (2010) advise omitting metformin on the day of procedure till the time oral intake is resumed. The British Diabetes Society guidelines allow metformin on the day of surgery in surgeries where the duration of fasting is limited to one meal. In patients with low glomerular filtration rate (GFR) and in those at risk of CIN, e.g., diabetics, it is advisable to withdraw metformin on the procedural day and restart after resuming oral intake and confirming stable renal function.

- **Thiazolidinediones:** Reduce peripheral insulin resistance and may reduce hepatic glucose production, e.g., rosiglitazone, pioglitazone. There are some concerns regarding increased cardiac events in patients taking rosiglitazone leading to its withdrawal from the market in some countries.
- **α -glucosidase inhibitors:** Suppress the breakdown of complex carbohydrates in the gut, delaying the postprandial rise in blood glucose concentration, e.g., acarbose.
- **Glucagon-like peptide-1 (GLP-1) analogs, e.g., liraglutin:** They are agonists at the GLP-1 receptor. GLP-1 is an intestinal hormone which exerts an insulinotropic action such as glucose-dependent stimulation of insulin and suppression of glucagon secretion.
- **Dipeptidyl peptidase-4 (DPP-4) inhibitors, e.g., sitagliptin, saxagliptin, vildagliptin, and alogliptin.** DPP-4 inhibitors prevent degradation of GLP-1, thereby improving blood sugar control. Combination therapy of DPP-4 with basal insulin has shown improved glycemic efficacy without increased rates of hypoglycemia. Linagliptin has the advantage of not needing dose correction with renal dysfunction.
- **Sodium-glucose cotransporter 2 (SGLT-2) inhibitors, e.g., dapagliflozin, canagliflozin, and empagliflozin.** These drugs oppose the action of SGLT-2 which is a glucose transporter present in the proximal tubule and is responsible for the reabsorption of most of the glucose in the kidney. Thus, they reduce hyperglycemia by promoting urinary glucose excretion. SGLT-2 inhibitors can cause euglycemic diabetic ketoacidosis (DKA) leading to severe acidemia. It may be advisable to stop the drug preferably 3 days prior to elective surgery and restarted 24 hours after resumption of oral intake.

Q. Which are the various types of insulins available?

Ans.

Insulin preparations: Extracted from beef (now rarely used) or pork pancreas—contained animal antigens which caused immunologic reactions and antibody formation.

Biosynthetic human-sequence insulins are synthesized using recombinant DNA technology from *Escherichia*

coli. The three types of insulin preparation are classified according to their length of action:

1. **Soluble insulins:** These have a rapid onset and short duration of action (depending upon the route of administration). When injected subcutaneously the duration of action is from 30 minutes up to 8 hours with a peak at 2–4 hours. When injected IV, it has a half-life of approximately 5 minutes. Insulin lispro and insulin aspart, which are recombinant human insulin analogs, have a faster and shorter action profile.
2. **Intermediate-acting preparations:** Made with suspensions of insulin with either protamine (isophane insulin) or zinc (crystalline insulin) salts or both together. They are administered in combination with soluble insulin to obtain rapid onset together with a long duration of action. Isophanes produce peak plasma insulin levels at variable intervals between 4 and 8 hours after injection, and their glucose-lowering action wears off rapidly after 10–12 hours.
3. **Longer-acting insulin preparations:** Peak effect occurring after 10–12 hours and its duration of action may be 16–18 hours. They are not suitable for IV use. Long-acting insulins may act for up to 36 hours for animal and 24 hours for human-sequence preparations.

Insulin supplementation: Physiologically, insulin secretion varies in response to feeding or starvation. A basal insulin level is maintained during the fasting state. These conditions are almost impossible to replicate with exogenous insulin administration. Consequently, insulin administered subcutaneously, even if timed optimally, will have inadequate peak concentrations and inadequate basal levels causing periods of hypoinsulinemia and hyperglycemia. Also, its entry into the systemic circulation rather than into the portal system affects its metabolic actions. Absorption of insulin injected subcutaneously is slow and unpredictable in the perioperative period and IV administration is advised.

Insulin regimes comprise of basal, prandial and/or corrective doses. The regime and dose will depend upon the preoperative diabetes control, oral intake, and presence of factors predisposing the patient to hyperglycemia.

Q. What are the newer insulins available and their anesthetic implications?

Ans. Recently some newer insulin preparations were marketed as long-acting insulin analogs. These are molecular engineered formulations designed to improve the pharmacokinetics, absorption profile, and duration of action of human insulin.

Glargine: It has onset of action of 2–4 hours and duration of action of 20–24 hours.

Detemir: It had onset of action of 1–4 hours and duration of action of 12–24 hours.

Degludec: Onset of action within 30–90 minutes with a duration of action >24 hours.

Degludec plus: Degludec combined with insulin aspart suspension, onset of action 5–15 minutes, peak action 30–60 minutes, and duration of action >24 hours.

Diabetic patients should be monitored for hypoglycemia in the perioperative period as hypoglycemia under anesthesia may be longer in duration and more severe. Newer insulin and its analogs, with longer duration of action may show hypoglycemia as an adverse effect, particularly, if used in conjunction with other oral or parenteral hypoglycemics. Decreasing the dose of premeal insulin or avoiding simultaneous oral hypoglycemics reduces the risk of hypoglycemia. These adverse effects may occur particularly in the first 4 weeks of treatment, and declines over time. New long-acting insulins, such as glargine and detemir, show good glycemic control between meals without the risk of hypoglycemia, so they can be used as a basal insulin during the perioperative period.

Future of Insulin Therapy

In 2006, an inhaled form of insulin (*Exubera*) was available but it was discontinued because it failed to gain broad acceptance by patients and clinicians. In June 2014, a new formulation *Afrezza* (human insulin) inhalation powder was introduced to cover prandial insulin requirements in nonsmoking adults with diabetes who are free of pulmonary disease.

Q. What is the principle of perioperative blood sugar control management?

Ans. The aim is to maintain patient's usual regime as far as possible to avoid disrupting the control. A short fasting period and early return to normal intake is essential for this approach. Stopping drugs increases the risk of hyperglycemia and catabolism whereas failure to establish feeding increases the risk of hypoglycemia. The plan should be decided after due consideration of risks and benefits.

Patients with good preoperative blood sugar control for elective surgery are continued on their regular oral antidiabetic treatment and regular (short-acting) insulins on the night prior to surgery. However, long-acting insulins may cause hypoglycemia in a fasting patient, so the night dose may be reduced. NPH insulin dose is reduced to 50% and that of newer agents such as glargine or detemir is reduced to 80% on the previous night. Blood glucose measurement should be done on morning of surgery and glucose supplementation given if required.

Q. How is the drug therapy managed on the day of surgery in patients who are expected to resume normal diet soon after surgery?

Ans. Secretagogues (e.g., sulfonylureas, meglitinides) and SGLT-2 inhibitors are omitted on the morning of surgery. DDP inhibitors are safe to be taken on the day of surgery. Thiazolidinediones can be given on morning of surgery if not more than one meal is likely to be missed but should be omitted otherwise. Acarbose is omitted when the patient is fasting. Metformin can be given on morning of surgery if a short fasting period is expected, patient has no renal dysfunction and the surgery is unlikely to cause hypotension and hypoperfusion.

Short-acting insulins are omitted during fasting. Some guidelines recommend giving reduced doses (one-third or half of usual dose) of intermediate to long-acting insulin on the morning of surgery with blood sugar monitoring, the rationale being to supplement basal insulin requirements and prevent ketoacidosis and catabolic state (particularly in patients with absolute insulin deficiency). Vigilance is required to avoid hypoglycemic episodes in these patients. Patients on insulin pumps should be continued on the smallest basal infusion rate.

Q. What are the implications of surgery in a diabetic patient?

Ans. The perioperative problems posed by surgery in the diabetic patient are:

- Stress response to surgery (magnitude depends on site of surgery, tissue injury, etc.):
 - The secretion of catecholamines, cortisol and growth hormone oppose glucose homeostasis as these hormones have “anti-insulin” and hyperglycemic effects. Glycogenolysis and gluconeogenesis are stimulated and peripheral glucose uptake is decreased causing hyperglycemia and ketosis. Perioperative insulin administration is required to control blood sugar.
 - Rise in blood sugar during surgery is related to magnitude of inflammation. Excessive release of inflammatory cytokines, such as tumor-necrosis factor—interleukin-1, and interleukin-6 and immobility itself is associated with reduced skeletal muscle insulin sensitivity leading to hyperglycemia.
 - In fasting patients undergoing elective intra-abdominal procedures, blood glucose levels typically increase to between 126 and 180 mg/dL. During cardiac surgery the disturbance of glucose homeostasis is greater, with blood glucose values rising above 270 mg/dL in subjects without diabetes and above 360 mg/dL in subjects with diabetes. Stress may precipitate diabetic crisis (ketoacidosis or hyperglycemic hyperosmolar coma).

- Interruption of oral intake, which may be further prolonged after gastrointestinal surgery. Starvation can lead to ketosis.
- Altered consciousness in perioperative period may mask the symptoms and signs of hypoglycemia.
- Circulatory disturbance associated with anesthesia for major surgery may alter the absorption of subcutaneous insulin. So IV administration is preferred intraoperatively.
- Hyperglycemia is associated with increased risk of complications such as wound infection and poor neurological outcomes in susceptible patients.

The goals for the diabetic patient are minimal metabolic disruption, avoidance of untoward events, and return to stable glycemic control as soon as possible.

Q. What are the adverse effects of hyperglycemia?

Ans. Poor glycemic control has been shown to increase the risk of poor wound healing, perioperative infections, and myocardial and renal dysfunction.

Acute consequences of untreated, or inadequately treated, DM include:

- Dehydration resulting from the osmotic diuretic effect of glycosuria.
- Acidemia because of accumulation of ketoacids and/or lactic acid.
- Electrolyte imbalance—potassium and magnesium imbalance—which can increase arrhythmia risk.
- Fatigue, weight loss and muscle wasting because of lipolysis and proteolysis in absolute insulin deficiency.

Q. How will you investigate a diabetic patient for surgery?

Ans.

Glycosylated hemoglobin (HbA1c): HbA1c of <7% (average blood sugar 150 mg%) implies good blood sugar control over the preceding 8–12 weeks (life span of RBC is 120 days). It is a weighted average with the blood sugar level in the preceding 30 days contributing more to the level.

One should consider delaying elective surgery in patients with HbA1c levels >8.5% (69 mmol/mol). This corresponds to an average blood sugar level of around 200 mg%.

Fasting and postprandial blood sugar: Have been traditionally used to assess quality of control. It is now accepted that HbA1c is superior to assess long-term control. Fasting value of 80–120 mg% and bedtime value of 100–140 mg% was regarded as optimal preoperative blood sugar control for elective surgeries.

Hemoglobin: Anemia may be seen in patients with renal insufficiency. It is also as a baseline investigation to guide blood transfusion intraoperatively.

White cell count: Look for infection (leukocytosis).

Urine routine for microalbuminuria: Alerts clinician to the presence of diabetic nephropathy and possibly other microvascular complications.

Serum creatinine: To detect renal dysfunction. Estimated GFR should be calculated.

Serum electrolytes: To detect abnormalities in patients with history of vomiting, diarrhea, poor oral intake or tube feeding, intestinal obstruction, etc. Also in patients on insulin, ACE inhibitors, diuretics, and renal dysfunction, serum potassium levels can be abnormal.

Electrocardiogram: To detect asymptomatic myocardial ischemia. Diabetic men are more than four times as likely, and women five times more likely to have coronary heart disease than nondiabetics.

2D echocardiography: It should be done (if facility available and time permits for an urgent surgery in a sick patient) for patients with long-standing diabetes and in those with cardiovascular symptoms.

(Noninvasive or invasive cardiac testing may be warranted preoperatively, particularly for the diabetic undergoing major noncardiac and vascular surgery).

X-ray chest: Routine X-ray chest is not recommended. X-ray chest may be prescribed in symptomatic patients and those at risk of respiratory problems. Tuberculosis is common in diabetics due to impaired immunity. Patients with abdominal distension may have pneumonia secondary to basal atelectasis or aspiration.

Morning of surgery investigations: Serum electrolytes, FBS.

Emergency surgery: Acute illness can cause metabolic decompensation. These patients need full clinical and biochemical assessment. Urine ketones are done for detection of ketonuria. More than 2+ on ketone dipsticks is significant. The most common cause of ketoacidosis is DKA. Occasionally, ketoacidosis may be due to starvation or alcohol ingestion.

Q. What are the principles of anesthesia management in a diabetic patient?

Ans.

Timing: Diabetic patients should be placed first on the operating list. This shortens their preoperative fast and the risk of hypoglycemia and ketosis.

Preoperative fasting: Delayed gastric emptying due to DAN is found in up to 50% of type 1 DM patients. Undiagnosed gastroparesis may prolong retention of food in the stomach thereby increasing the risk of regurgitation and aspiration. Some studies have shown beneficial effects of preoperative

oral erythromycin on gastric motility. However, other motility stimulants have not been shown to be effective. A 12-hour fast may be beneficial in diabetic patients with suspected gastroparesis. A prolonged fasting increases catabolism and increases insulin resistance, so patients with long fasting times should receive a glucose insulin infusion.

Intravenous fluids: Ringers lactate is generally considered safe in diabetic patients. Lactate undergoes gluconeogenesis in the liver and may cause hyperglycemia when given rapidly in large volumes. Normal saline infusions carry the risk of hyperchloremic acidosis. Thus, there is no ideal solution.

Ringer's lactate can be safely used in most cases with monitoring of blood sugar level. Balanced salt solutions containing acetate and gluconate, when compared with normal saline have been shown to reduce acidosis in the early management of DKA though their impact on perioperative glucose management is not clear.

Intraoperative monitoring: Frequent, rapid, and accurate blood glucose measurement is essential in the anesthetized patient as the requirements for glucose and insulin in this period are unpredictable and hypoglycemia may go undetected. A 1–2 hourly monitoring is advisable in patients receiving insulin. A 4 hourly monitoring is sufficient in well-controlled diabetics on OHA.

Standard monitoring: ECG, SpO_2 , BP, ETCO_2 , and temperature should be instituted. Advanced monitoring is guided by presence of comorbidities.

Glycosylated hemoglobin measurement has no value in the intra- or postoperative period but is a valuable guide to long-term glycemic control during preoperative evaluation.

Sugar control: "Permissive hyperglycemia" is unacceptable in current practice with availability of more accurate, rapid and easy-to-use capillary blood glucose monitors. It is recommended to maintain blood sugars between 110–180 mg%. Postoperative wound healing and infection may be influenced by the adequacy of perioperative glycemic control. Similarly blood sugars < 70 mg% are associated increased postoperative morbidity and mortality.

Intraoperative Short-acting Insulin Infusion

It is essential in patients with absolute insulin deficiency to prevent lipolysis and proteolysis with resultant ketosis. Other patients who require insulin infusion intraoperatively are patients with poorly controlled diabetes, those who are unlikely to resume normal oral intake after surgery, and patients with infections. Insulin infusion rate is titrated to target blood sugar levels.

These patients should receive substrate in the form of glucose. Hypotonic solutions like 5% dextrose are undesirable due to the risk of hyponatremia. 5% dextrose in 0.45% NaCl with 20 mmol of potassium/L is the preferred solution for maintenance fluid therapy (1–2 mL/kg/h) in patients who require insulin infusions intraoperatively. Ringer's lactate solution is used for replacing fluid deficits.

Patients who cannot be established on enteral feeding soon after surgery should receive this regime postoperatively. Balanced solutions such as Ringer's lactate solution are given for additional intravascular volume requirements.

Glucose Supplementation

Routine administration of glucose-containing solutions is not recommended in patients not needing insulin infusions. Diabetic patients receiving longer-acting insulin should have their blood sugar levels monitored and may require glucose infusions as they are at risk of hypoglycemia if glucose is not supplemented. Perioperative administration of glucose enhances postoperative glucose utilization rates and prevents excessive catabolism and starvation ketosis.

Anesthesia

Induction: Choice of agent for general anesthesia depends on severity of systemic diseases, such as coronary artery disease, nephropathy, hypertension, and autonomic neuropathy. Epidural analgesia may be instituted after due consideration to autonomic neuropathy, ischemic heart disease (IHD), and peripheral neuropathy. It should be avoided in sick patients with sepsis. Epidural analgesia may help to attenuate neurohormonal response to stress and avoid systemic analgesics such as nonsteroidal anti-inflammatory drugs (NSAIDs) and opioids which may have serious side effects in a diabetic patient. A rapid sequence induction should be performed for patients with GI symptoms. Awake fiber-optic bronchoscopy would be preferred technique for an anticipated difficult airway.

A careful induction with etomidate or high-dose fentanyl (4–5 $\mu\text{g/kg}$) with midazolam and/or thiopentone should be performed in patients with cardiovascular complications (exaggerated hypotension due to autonomic neuropathy is common). Succinylcholine should be avoided in patients with extensive peripheral neuropathy due to risk of increased potassium release. Atracurium and mivacurium are preferred in presence of renal dysfunction. Rocuronium may be used in rapid sequence induction. Anesthesia is maintained with isoflurane or sevoflurane in an air oxygen/oxygen-nitrous oxide mixture.

Hypotension may be hazardous in diabetic patients due to presence of angiopathies which increase the risk of critical hypoperfusion of the heart, brain, and kidney, and should be promptly treated.

Q. Do anesthetic drugs affect blood sugar control?

Ans.

Induction agents: Ketamine may cause significant hyperglycemia. Etomidate blocks adrenal steroidogenesis and hence cortisol synthesis and decreases the hyperglycemic response to surgery by approximately 18 mg% in nondiabetic subjects. The effects on diabetic patients have not been established. The effect of propofol on insulin secretion is not known. Diabetic patients show a reduced ability to clear lipids from the circulation. This should be considered in patients receiving propofol for prolonged sedation in the intensive care unit.

Halothane, enflurane, isoflurane, and sevoflurane in *in vitro* studies inhibit the insulin response to glucose in a reversible and dose-dependent manner but their effect in clinical situations is not certain.

Benzodiazepines decrease the secretion of adrenocorticotrophic hormone (ACTH) and the production of cortisol, when used in high doses. They reduce sympathetic stimulation but stimulate growth hormone secretion and result in a decrease in the glycemic response to surgery. These effects are minimal when midazolam is given in usual sedative doses.

High-dose opiate anesthetic techniques produce hemodynamic, hormonal and metabolic stability. These techniques effectively block the entire sympathetic nervous system and the hypothalamic-pituitary axis, probably by a direct effect on the hypothalamus and higher centers. However, midazolam and fentanyl may cause hyperglycemia by reducing glucose clearance.

Ganglion-blocking agents (used for hypotensive anesthesia previously) may block sympathetically mediated hepatic gluconeogenesis with resultant hypoglycemia. The use of β -blockers is associated with slower recovery from hypoglycemia.

Q. Describe hyperglycemic emergencies.

Ans.

- Diabetic ketoacidosis
- Hyperosmolar hyperglycemic state (HHS).

The underlying mechanism for both disorders is a reduction in the action of circulating insulin along with a rise of counter regulatory hormones, such as glucagon, catecholamines, cortisol, and growth hormone. DKA is associated with absolute deficiency of insulin whereas HHS is seen with lesser degrees of insulin deficiency.

Increased gluconeogenesis and hepatic and renal glucose production along with impaired glucose utilization in peripheral tissues cause hyperglycemia and hyperosmolality. In DKA, the increased counter regulatory hormones lead to lipolysis and hepatic fatty acid oxidation to ketone bodies (hydroxybutyrate, acetone, and acetoacetic

acid) with resulting metabolic acidosis. Osmotic diuresis leads to water loss and electrolyte disturbance.

Diabetic Ketoacidosis

It is seen in type 1 diabetics and patients with type 2 diabetes during the catabolic stress of acute illness such as trauma, surgery, or infection. DKA is sometimes the presenting feature of type 1 diabetes. DKA is sometimes seen due to discontinuation of antidiabetic treatment or with drugs that affect carbohydrate metabolism such as corticosteroids, thiazides, sympathomimetic agents and some antipsychotics agents. DKA is a proinflammatory state producing reactive oxygen species, proinflammatory cytokines, plasminogen activator inhibitor-1, and C-reactive protein.

The clinical features are:

- Symptoms of hyperglycemia such as polyuria and polydipsia.
- Symptoms of acidosis and dehydration such as respiratory distress, drowsiness, coma, and abdominal pain.
- Other symptoms such as vomiting, malaise, cramps, and signs of infection.
- Signs include tachycardia, dehydration, acidotic breathing, depressed consciousness, and abdominal tenderness.

The classical triad of DKA is ketonemia, hyperglycemia, and metabolic acidosis.

The Joint British Diabetes Societies for Inpatient Care group has defined DKA as ketonemia > 3 mmol/L or ketonuria $> 2+$ by dipstick method, blood sugar > 11 mmol/L (200 mg%) or known DM, and venous pH < 7.3 or serum bicarbonate < 15 mmol/L.

Resolution of DKA is defined as serum bicarbonate > 15 mmol/L, ketonemia < 0.6 mmol/L and venous pH > 7.3 .

Hyperosmolar Hyperglycemic State

Hyperosmolar hyperglycemic state is seen in type 2 diabetics, more commonly in elderly people with coexisting diseases. HHS may be caused by plasma insulin concentrations that are inadequate for glucose utilization but are adequate to prevent lipolysis and ketogenesis. The hyperglycemia and volume deficit is more severe than DKA.

Clinical Features

- Slow onset over days to weeks
- Symptoms of hyperglycemia
- Signs of dehydration
- Signs of hyperviscosity and thrombosis such as delirium, coma, seizures, sensory, and motor deficits

- Abdominal pain is unusual. Vomiting may be present. Investigations for diagnosis in both DKA and HHS include.

Complete blood count (CBC), arterial blood gas (ABG), serum glucose, urine analysis, electrolytes, renal and liver biochemistry, ECG, X-ray chest, and appropriate cultures (Table 1).

TABLE 1: Typical values and total body deficits of water and electrolytes in DKA and HHS.

	DKA	Severe DKA	HHS
Plasma glucose mmol/L (~ mg%)	>11 (200)		>30 (540)
Arterial pH	<7.3	<7.0, anion gap > 16	>7.3
Serum bicarbonate (mEq/L)	<15	<5	>15
Urine ketone	++ (on dip stick)		Small
Serum ketone (mmol/L)	>3	>6	Small
Serum osmolality	Variable		>320 mOsm/kg
Mental status	Alert/drowsy/stupor/coma based on severity	GCS < 12	Stupor/coma
Water deficit (mL/kg)	100	SBP < 90 mm Hg, PR < 60, >100	100–200
Na deficit (mEq/kg)	7–10		5–13
Cl deficit (mEq/kg)	3–5		5–15
K deficit (mEq/kg)	3–5	Serum K < 3.5 mmol/L	4–6
PO ₄ deficit (mEq/kg)	5–7		3–7
Mg deficit (mEq/kg)	1–2		1–2
Ca deficit (mEq/kg)	1–2		1–2

(DKA: diabetic ketoacidosis; GCS: Glasgow coma scale; HHS: hyperosmolar hyperglycemic state; PR: pulse rate; SBP: systolic blood pressure).

Q. How would you treat DKA and HHS?

Ans. Principles of treatment of DKA and HHS:

- Correction of dehydration, hyperglycemia, and electrolyte imbalances
- Identification of comorbid precipitating events
- Frequent patient monitoring.

Intensive fluid resuscitation is vital to the management in both cases.

In DKA, the focus is on clearing the ketones and controlling the blood sugar. The targets for treatment are average hourly improvement as follows: ketonemia by 0.5 mmol/L, glucose by 3 mmol/L (~50 mg%), bicarbonate by 3 mmol/L with potassium between 4 and 5.5 mmol/L.

In HHS, correction of high osmolality without causing sudden drops, and correction of fluid, and electrolyte deficit is most important. Insulin is recommended at lower doses compared to DKA to prevent rapid drop in blood sugar level and osmolality, and is started after fluid resuscitation.

Fluid therapy: The aim of fluid therapy is expansion of the intravascular and extravascular volume, and restoration of renal perfusion. Normal saline is fluid of choice for initial resuscitation in HHS to correct sodium deficit and to prevent sudden drop in osmolality. Normal saline is also the recommended IV fluid in DKA though balanced salt solutions such as Ringers lactate have also been used in the early resuscitation of DKA with some studies showing better acidosis improvement in the initial period. Colloids are not recommended.

Rate of fluid therapy: 15–20 mL/kg body weight is infused in the first hour. The rate is halved over the next 4 hours. Further fluid resuscitation is guided by assessment of fluid status, urine output and electrolyte level. Total fluid deficit is corrected over 24 hours with constant monitoring to avoid fluid overload.

Children and young adults have higher risk of cerebral edema and fluid therapy has to be guarded, particularly in patients who do not present in shock. Also fluid therapy has to be guarded in the elderly, and pregnant patients, and those with significant comorbidities such as cardiac and renal insufficiency.

Patients in hemodynamic shock need fluid and vasopressor therapy, and advanced hemodynamic monitoring.

Insulin Therapy

Diabetic ketoacidosis: Fixed rate continuous infusion at a dose of 0.1 unit/kg/h should be administered with the aim to decrease plasma glucose concentration at a rate of ~50 mg%/h. If the drop is not adequate, insulin dose can be increased to achieve a steady drop. When blood sugar drops to below 250 mg%, dextrose is added to allow continuation of fixed rate insulin infusion as insulin is required for clearing of ketones.

Hyperosmolar hyperglycemic state: Fluid resuscitation itself causes a drop in glucose levels. Insulin is started after initial fluid resuscitation and at rates of 0.05 units/kg/h with target drop in blood glucose of 4–6 mmol/L (~70–100 mg%). Serum sodium should not drop by >10 mmol/L in 24 hours.

Potassium therapy: Potassium supplementation is avoided for levels >5.5 mEq/L or in presence of reduced urine output. All other patients should receive potassium. For serum potassium <3.3 mEq/L, potassium supplementation is started before starting insulin to avoid arrhythmias, cardiac arrest or respiratory muscle weakness.

Bicarbonate: Use of bicarbonate in DKA is controversial with no universally followed threshold for administration. Bicarbonate is usually reserved for severe metabolic acidosis with $\text{pH} < 7.0$.

About 50–100 mmol/L bicarbonate can be given depending on the severity of acidosis. However, bicarbonates should not be used in presence of hypokalemia till serum potassium levels are corrected. Close monitoring of blood potassium levels and aggressive correction is advised at serum potassium levels <3.3 mEq/L.

Phosphate: Supplementation is advised only at serum phosphate concentration <1.0 mg/dL or in the presence of anemia, cardiac dysfunction, or respiratory muscle weakness. If needed, 20–30 mEq/L potassium phosphate can be given. Routine use of phosphate is not encouraged.

Ketone bodies: The commonly used nitroprusside method measures only acetoacetic acid and acetone and not β -hydroxybutyrate which is the strongest and most prevalent acid in DKA. Also during treatment, β -hydroxybutyrate gets converted to acetoacetic acid making it appear that the ketosis has worsened if the nitroprusside method is used. Therefore, hydroxybutyric acid monitoring is preferred and new bedside monitors are now available.

Monitoring: 2–4 hourly estimation of serum electrolytes, glucose, blood urea nitrogen, creatinine, osmolality, and acid-base status.

Patient can be started on a new regime or his previous subcutaneous regime, after DKA resolves.

In HHS, serum glucose is maintained between 250 and 300 mg % till the osmolality is <315 mOsm/L and the patient is mentally alert.

Complications

- Cerebral edema:
 - Most dangerous complication with high mortality rate
 - Could be related to rapid drop in plasma osmolality or to increased cerebral perfusion.
- Iatrogenic hypoglycemia and hypokalemia.
- Pulmonary edema and hypoxemia.

Q. What are the specific things that you will consider in a diabetic patient who develops intestinal obstruction for emergency laparotomy?

Ans.

• Intestinal obstruction:

- Fluid and electrolyte status:
 - Signs of hypovolemia/dehydration—tachycardia, low volume pulse, low jugular venous pulse (JVP), hypotension, dry tongue, low urine output—these patients will need resuscitation prior to anesthesia.
 - Has the patient been vomiting/have high nasogastric aspirate/severe abdominal distension?—He may have electrolyte and acid base abnormality; evaluate ABG and serum electrolytes.
- Signs of peritonitis/sepsis—fever, abdominal guarding:
 - Prompt broad spectrum antibiotics
 - Resuscitation based on “early goal directed therapy.”

• Diabetes related concerns:

- How long has the patient been unable to take orally?
- Has the patient been taking OHA/insulin? (Dose and time).

Patient is at risk of developing DKA especially if he has been unable to take insulin or has infection; starvation ketosis if he has not been feeding or hypoglycemia if he is on long-acting antidiabetic medications.

Measurement of blood sugar, urine ketones and in some cases ABG (for metabolic acidosis) is indicated.

Q. The surgeon has booked a diabetic patient for an emergency laparotomy for perforative peritonitis. On questioning, he says the patient is stable, has a normal blood pressure and he wants to start immediately before the patient starts worsening. You find the patient has a PR 120/min, BP 100/70 mm Hg and is drowsy. What would your management plan be?

Ans. Drowsiness in this patient is most probably metabolic in origin with the following differential diagnosis:

- Sepsis
- Diabetic ketoacidosis
- Hypoglycemia
- Electrolyte (sodium) imbalance
- Hypoperfusion due to cardiac dysfunction or hypovolemia
- Hypoxemia
- Uremia
- Hyperosmolar hyperglycemic state
- Sedative medications.

Tachycardia is likely to be due to hypovolemia and low stroke volume (despite a “normal” blood pressure). This patient should be optimized prior to surgery. The

patient may have a cardiovascular collapse if anesthesia is induced in this condition. Optimization will include fluid resuscitation, antibiotic administration, insulin/glucose administration and/or vasoactive drugs according to the clinical condition. Invasive monitoring—intra-arterial BP and CVP—should be instituted.

Investigations for Diagnosis and Monitoring

- Immediate bedside blood sugar estimation
- Urine ketones
- Oxygen saturation monitoring
- ABG with serum electrolytes and lactates
- Detailed history (fever, etc.) and drug chart examination
- Fluid balance (intake/output) since admission
- Serum biochemistry.

Acute abdominal pain in a diabetic patient should be evaluated in detail. Diabetes-related complications such as DKA, gastroparesis, and intractable vomiting can lead to a false diagnosis of acute surgical emergency. Diabetic pseudo-tabes syndrome is a rare syndrome characterized by sharp neuropathic pain along thoracolumbar dermatomes which can be confused with visceral disorders. These patients have pupillary and gait abnormalities from associated cranial and peripheral neuropathy.

Q. How will you optimize this patient?

Ans.

- Fluid resuscitation:
 - Fluid administration should be started immediately in hypovolemic patients through wide-bore cannulae. Normal saline/Ringer's lactate in a volume of 5–20 mL/kg may be given depending on clinical assessment of fluid status (In patients with cardiac dysfunction, fluid boluses should be given cautiously).
 - In sick patients, a goal directed approach for resuscitation has been shown to improve outcomes. However, the goal to be achieved is controversial. Older studies advocated a central venous oxygen saturation of 70–75%. Many recent studies have used stroke volume or stroke volume-based targets. These require use of advanced hemodynamic monitors such as esophageal Doppler monitor, arterial waveform-based cardiac output monitor, etc. Central line may be useful for administration of vasoactive drugs but its use for assessing volume responsiveness is limited.
- Continuous intra-arterial BP monitoring. Vasopressors and inotropes may be required to optimize cardiac output.
- Appropriate broad-spectrum antibiotics should be administered before incision.

- Oxygen supplementation to increase oxygen delivery. Intubation and mechanical ventilation may be required in the severely decompensated patients to reduce work of breathing and improve oxygen supply demand balance.
- IV insulin infusion should be started to control blood sugars. 5% dextrose in normal saline should be given as maintenance fluid at the rate of 2 mL/kg/h, apart from resuscitation fluids.

Adsorption of insulin on to the surface of syringes, IV fluid bags and IV infusion sets is an unavoidable problem. Adding albumin or a few milliliters of patient's blood to the infusate was previously practiced to reduce this effect. Flushing around 50 mL of solution is also thought to be effective. In solutions with a concentration of insulin of >10 U/L, the effect is minimal. More consistent delivery can be achieved with more concentrated solutions of lower volume administered from a syringe.

Q. How will you manage perioperative blood sugar control?

Ans. Aims of perioperative management of diabetes are:

- Avoiding hyperglycemia
- Avoiding ketosis
- Avoiding hypoglycemia.

All elective cases should have good long-term control as assessed by HbA1c. Insulin-treated patients undergoing major elective surgery may require admission before surgery if blood sugar control is suboptimal. Intraoperatively, the upper limit of acceptable blood sugar level is controversial. Most problems are noted above blood sugar levels of 180 mg% but there might be advantages of tighter sugar control in some patient groups. Traditionally, blood sugars were maintained in the higher range due to the fear of hypoglycemia. Now, with increasing evidence of the risks of hyperglycemia as well as easily available bedside glucose tests, permissive hyperglycemia is no longer acceptable. Similarly overenthusiastic blood sugar control can lead to hypoglycemia which is also deleterious. Regular blood sugar measurement is important to prevent catastrophic consequences of extremes of glycemia.

Current guidelines recommend use of variable rate IV insulin infusions with maintenance glucose supplementation intraoperatively and postoperatively whenever intervention is required for blood sugar control. In minor surgery however, perioperative hyperglycemia (>200 mg/dL) may be managed with a small subcutaneous dose of short-acting insulin with frequent monitoring to avoid hypoglycemia. Glucose-insulin-potassium (GIK) infusions may be in use in some institutes in the intraoperative period.

Standard GIK Solution

- 500 mL 10% dextrose solution + 15 units short-acting insulin + 10 mmol KCl
- Infuse over 5 hours (100 mL/h)
- 5% dextrose solutions are used by some instead of 10% dextrose
- Short-acting insulin is added at 0.32 units/g of dextrose. Potassium is avoided in patients with renal dysfunction and those with hyperkalemia.

Advantages

- Inherently safe with separate glucose and insulin infusions, one may be stopped inadvertently with potentially harmful consequences of hypoglycemia or hyperglycemia.
- Do not need sophisticated equipment such as infusion pumps.

Disadvantages

- Insulin depends on receptor sensitivity for its action in vivo. Therefore, adjustments have to be made to insulin dose to be added to "neutralize" the dextrose which may involve making fresh solutions.
- Fine control of sugar control more difficult. Separate infusions are easier to titrate the blood sugar levels.

Separate Infusions

The obligate glucose requirement to provide for cells which can only use glucose as substrate such as neurons and RBCs is 2 mg/kg/min. This can be provided by giving 5% or 10% solution. 5% dextrose solutions are given at the rate of 100–125 mL/h. The initial insulin infusion rate can be estimated as between one-half and three-fourths of the patient's calculated hourly insulin requirement [patients total daily dose (TDD) divided by 24]. Insulin requirements are higher in septic, obese, critically ill patients, and in those receiving steroids or undergoing cardiopulmonary bypass surgery.

Q. How will you control blood sugars intraoperatively?

Ans. Target and management of blood sugar intraoperatively depend upon the duration of surgery, type of surgery, type of anesthesia, preoperative antidiabetic therapy, and expected time to resume oral intake after surgery. The Endocrine Society and Society of Ambulatory Anesthesia recommend that intraoperative blood glucose levels be maintained <180 mg/dL. Hyperglycemia (>180 mg/dL or 10 mmol/L) is treated with subcutaneous (SC) rapid-acting insulin analogs or with an IV infusion of regular insulin. Patients undergoing ambulatory surgery or short surgical procedure (<4 hours operating room time) or minimally invasive surgery procedures can be managed effectively by SC insulin treatment and allow early resumption of oral intake.

If SC insulin is used preoperatively or intraoperatively for blood sugar control, blood glucose testing is required at least every 2 hours.

Correctional insulin (rapid-acting insulin) is defined as the supplemental insulin administration for blood glucose > 180 mg/dL (10 mmol/L) following blood glucose testing.

$$\text{Correctional dose of insulin} = \frac{\text{Measured glucose} - 100}{\text{insulin sensitivity factor}}$$

Insulin sensitivity factor is equal to 1,800 divided by the patient's TDD of insulin. The TDD is equivalent to the patient's daily amount of basal, prandial, and correctional insulin. If TDD is not known or if a patient is on oral antidiabetic medications, a sensitivity factor (denominator) of 40 provides a safe calculation for a correctional insulin dose (**Table 2**).

Rapid-acting insulin should not be repeated before 2 hours to minimize the risk of hypoglycemia.

An IV insulin infusion is recommended in patients undergoing procedures with anticipated hemodynamic changes, significant fluid shifts, expected changes in temperature [passive hypothermia or active cooling, hyperthermic intraperitoneal chemotherapy (HIPEC)], use of inotropes, or prolonged surgery (>4 hours). Absorption and distribution of SC insulin in these patients and conditions is unreliable which can lead to swings of hyperglycemia and hypoglycemia. Intravenous insulin infusions are also advisable in critically ill patients or during cardiac surgery. The short half-life of IV insulin (<15 minutes) allows for rapid-adjustment in drug delivery and has limited lasting effect. The target blood glucose in these type of surgeries is 140–180 mg/dL (**Table 3**).

TABLE 2: Correction SC insulin dosing regimens to treat intraoperative and postoperative hyperglycemia.

Blood glucose [mg/dL (mmol/L)]	Insulin sensitive (Age > 70, GFR < 45 mL/min, no history of diabetes)	Usual insulin	Insulin resistant (BMI > 35 kg/m ² , home TDD insulin > 80 U, steroids > 20 mg prednisolone daily)
141–180 (7.7–10)	0	2	3
181–230 (10–12.2)	2	3	4
231–260 (12.2–14.4)	3	4	5
261–300 (14.4–16.6)	4	6	8
301–350 (16.6–19.4)	5	8	10
351–400 (19.4–22.2)	6	10	12
>400 (>22.2)	8	12	14

Resumption of preoperative regime: May be delayed by factors such as a continuation of the stress response to surgery, persistent alterations in nutritional intake and the administration of drugs which may either reduce

TABLE 3: Infusion rate for continuous infusion of insulin during major surgeries.

Blood glucose level mg/dL (mmol/L)	If blood glucose increased from previous measurement	If blood glucose decreased by <30 mg/dL from previous measurement	If blood glucose decreased by >30 mg/dL from previous measurement
>241 (13.4)	Increase rate by 3 U/hour	Increase rate by 3 U/hour	No change in infusion rate
211–240 (11.7–13.4)	Increase rate by 2 U/hour	Increase rate by 2 U/hour	No change in infusion rate
181–210 (10–11.7)	Increase rate by 1 U/hour	Increase rate by 1 U/hour	No change in infusion rate
141–180 (7.8–10)	No change in infusion rate	No change in infusion rate	No change in infusion rate
110–140 (6.1–7.8)	No change in infusion rate	Decrease rate by 0.5 U/hour	Hold insulin infusion
100–109 (5.5–6.1)	- Hold the insulin infusion - Resume blood glucose measurement hourly - Restart insulin infusion at half the rate of initial infusion if blood glucose > 180 mg/dL (10 mmol/L)		
71–99 (3.9–5.5)	- Hold the insulin infusion - Repeat measurement every 30 minutes till blood glucose > 100 mg/dL (5.5 mmol/L) - Resume blood glucose measurement hourly - Restart insulin infusion at half the rate of initial infusion if blood glucose > 180 mg/dL (10 mmol/L)		
70 (3.9) or lower	If blood glucose 50–70 mg/dL (2.8–3.9 mmol/L): - Administer 25 mL of 50% dextrose - Repeat measurement every 30 minutes till blood glucose > 100 mg/dL (5.5 mmol/L) If blood glucose < 50 mg/dL (2.8 mmol/L): - Administer 50 mL of 50% dextrose - Repeat measurement every 15 minutes till blood glucose > 70 mg/dL (3.9 mmol/L) - When blood glucose > 70 mg/dL (3.9 mmol/L), repeat measurement every 30 minutes till blood glucose > 100 mg/dL (5.5 mmol/L) - Repeat 50 mL 50% dextrose if blood glucose < 50 mg/dL (2.8 mmol/L) second time and start 10% dextrose infusion - After blood glucose > 100 mg/dL (5.5 mmol/L) resume measurement hourly		

residual endogenous insulin secretion (in type 2 DM) or reduce insulin sensitivity. For minor surgeries under local anesthesia, e.g., cataract surgery, it is acceptable to allow normal oral intake and antidiabetic therapy throughout the perioperative period. If general anesthesia becomes necessary, the surgery is scheduled at a later date.

In pancreatic diabetes, hypoglycemia is more common and shows slow recovery due to low glucagon levels. These patients need more frequent blood sugar monitoring.

Q. What are the concerns with extubation in a patient with perforative peritonitis?

Ans. Patients with perforative peritonitis may benefit from postoperative ventilation for the following reasons:

- Bowel distension and a tense abdominal closure can cause splinting of diaphragm which reduces lung compliance and increases work of breathing. Airway pressures after abdominal closure should be observed to decide about need for postoperative ventilation.
- It can also lead to basal atelectasis leading to shunt and hypoxemia.
- Mechanical ventilation reduces oxygen demand of the respiratory muscle thereby optimizing oxygen delivery to vital organs in conditions of severe sepsis.

Less severe cases can be “reversed” and extubated at the end of surgery. The patient should have adequate recovery of airway reflexes prior to extubation.

Q. What is tight sugar control?

Ans. “Tight sugar control” refers to maintenance of blood sugar within a narrow normal range, typically between 70 and 110 mg%. However, hypoglycemia is a significant risk in this approach and therefore is not generally recommended. In some groups of patients, there is better evidence on the beneficial effects of tighter blood sugar control such as cardiac surgery, neuronal injury (e.g., traumatic brain injury, acute stroke), burns, transplant surgery, critically-ill patients and pregnant women.

Adverse neurologic outcome related to hyperglycemia is due to intracellular acidosis and a worsening of the hypoxic neuronal edema. Chemotaxis, phagocytosis, polymorphonuclear (PMN) adherence and apoptosis have been shown to be impaired by diabetes and hyperglycemia, increasing the risk of infection. Even glucose levels of 200 mg% may impair these defense mechanisms. Studies have generally shown benefit when glucose targets are in the broad range of 80–180 mg%.

In cardiac surgery, infection risk is higher in patients with poor postoperative blood sugar control, diabetic and nondiabetic. The data is less convincing for intraoperative and long-term glucose control. There is also evidence that overzealous intraoperative glucose control may be neurologically detrimental.

Q. Which factors influence the use of regional techniques in diabetic patients?

Ans.

Advantages of regional anesthesia:

- Compared to general anesthesia, epidural anesthesia has minimal effect on glucose metabolism. Circulating glucose, noradrenaline, and cortisol concentrations are not raised in patients undergoing epidural anesthesia for pelvic or lower limb surgery. Insulin response to a bolus of glucose is preserved in low spinal anesthesia (At high spinal levels, insulin secretions may be impaired due to sympathetic blockade). However, there is no hard evidence that choice of regional anesthesia and/or general anesthesia confers any benefit in terms of mortality and major complications.
- Patients are awake, so hypoglycemia and hyperglycemic coma is detected early.
- Allows early return to normal oral intake and resumption of regular treatment regime.
- Regional anesthesia also avoids the potential problems of tracheal intubation.
- Excellent analgesia. Spares use of systemic analgesics such as NSAIDs which can be deleterious in patients with diabetic nephropathy.

Disadvantages of regional anesthesia:

- Higher risk of nerve damage. Also ischemic nerve injuries are more likely with use of epinephrine in regional anesthesia in diabetics.
- Epidural anesthesia is less effective in reducing stress response in upper abdominal or thoracic surgery due to persistence of vagal afferent input.
- May carry higher risks in the diabetic patient with autonomic neuropathy. Severe hypotension may occur due to compromised cardiovascular compensatory mechanisms with deleterious consequences in a patient with coexisting coronary artery, cerebrovascular or renovascular insufficiency.
- The risks of infection and vascular damage may be increased with the use of regional techniques in diabetic patient.
- Epidural abscesses occur more commonly following spinal and epidural anesthesia in diabetic patients.
- Diabetic peripheral neuropathy presenting after regional anesthesia can be confused with an anesthetic complication of nerve block and can pose medicolegal problems (A detailed preoperative neurological examination, meticulous documentation of pre-existing neurological deficits and patient counseling is therefore essential).

(Note: Local anesthetic requirements may be lower in diabetic patients).

Q. How does open heart surgery influence blood sugar control?

Ans. Insulin requirements are increased with cardio-pulmonary bypass. The reason for the high insulin requirements in diabetics is due to insulin resistance caused by hypothermia, infusion of dead space of the bypass machine with glucose solution, and the hyperglycemic effects of adrenergic drugs.

Hyperglycemic hyperosmolar coma is known in patients undergoing cardiac surgery and carries high mortality.

Tight sugar control has been shown to reduce risk of postoperative infections.

Q. What is gestational diabetes mellitus?

Ans. Gestational diabetes mellitus (GDM) is defined as any presence of glucose intolerance with onset or first recognition during pregnancy.

Q. Explain pathophysiology of GDM.

Ans. The physiological changes of pregnancy are aimed to provide adequate nutrients to the growing fetus. In early pregnancy, maternal estrogen and progesterone increase and promote pancreatic β -cell hyperplasia and increased insulin release. Increases in peripheral glucose utilization and glycogen storage with a concomitant reduction in hepatic glucose production result in lower maternal fasting glucose levels. As pregnancy progresses, increased levels of human chorionic somatomammotropin, cortisol, prolactin, progesterone, and estrogen lead to insulin resistance in peripheral tissues. Cortisol has the highest diabetogenic potency and has peak effect at 26 weeks gestation. Progesterone also has relatively strong anti-insulin properties that peak at 32 weeks gestation. So tests for diagnosis are focused at these stages of pregnancy.

The mechanism of insulin resistance is likely a postreceptor defect. The pancreas releases 1.5–2.5 times more insulin in order to respond to insulin resistance. GDM results when there is delayed or insufficient insulin secretion in the presence of increasing peripheral resistance.

Independent risk factors for gestational diabetes:

- Body mass index above 30 kg/m^2
- Previous macrosomic baby weighing 4.5 kg or above
- Previous gestational diabetes
- Family history of diabetes
- Ethnicity—South Asian, Black Caribbean, Middle Eastern.

Q. What are the diagnostic criteria for GDM?

Ans. Screening of GDM is recommended at 24–48 weeks in women who were not previously diagnosed with overt diabetes. One-step or two-step screening test can be performed.

One-step Screening Test

In the morning, after at least 8 hours of fasting, 75 g oral glucose tolerance test (OGTT) with plasma glucose measurement is performed. This test should be repeated at 1 and 2 hours after initial measurement. Diagnosis is confirmed when plasma glucose is equal to or more than:

- Fasting: 92 mg/dL
- 1 hour: 180 mg/dL
- 2 hours: 153 mg/dL.

Two-step Screening Test

In first step of this test, perform a 50 g nonfasting glucose tolerance test with plasma glucose measurement at 1 hour. If plasma glucose after 1 hour is >140 mg/dL, perform the step two.

In step two (if plasma glucose in first step is >140 mg/dL) 100 g of OGTT is performed while patient is fasting. Diagnosis of GDM is confirmed if two or more plasma glucose levels are equal to or more than:

- Fasting: 95 mg/dL
- 1 hour: 180 mg/dL
- 2 hours: 155 mg/dL
- 3 hours: 140 mg/dL.

Q. How would you classify GDM?

Ans. White classification, named after Priscilla White who pioneered research on the effect of diabetes types on perinatal outcome, is widely used to assess maternal and fetal risk.

Gestational diabetes (diabetes which began during pregnancy).

Pregestational diabetes (diabetes that existed prior to pregnancy).

There are two subtypes of gestational diabetes:

Type A1: Abnormal OGTT but normal blood glucose levels during fasting and 2 hours after meals; diet modification is sufficient to control glucose levels.

Type A2: Abnormal OGTT compounded by abnormal glucose levels during fasting and/or after meals; additional therapy with insulin or other medications is required.

These two groups are further subdivided according to their associated risks and management.

Q. What preconception care is needed for diabetic women?

Ans. Good glycemic control before conception (HbA1c < 10%) and during pregnancy is important to reduce the risk of miscarriage, congenital malformation, stillbirth and neonatal death. Maintaining the HbA1c below 6.1% helps to reduce the risk of congenital malformations.

Metformin can be continued during pregnancy, but all other oral hypoglycemic agents should be stopped.

The primary concern is the drug crossing the placental barrier causing hyperinsulinemic hypoglycemia in the fetus. There were also some concerns regarding congenital malformations. Insulin is therefore preferred (recently there is evidence for efficacy and safety of glibenclamide and glyburide).

Rapid-acting insulin analogs (aspart and lispro) are safe for the fetus. Among the long-acting insulins, isophane insulin (NPH insulin) is preferred.

Angiotensin-converting enzyme inhibitors, angiotensin-II receptor antagonists and statins should be discontinued in the perigestational period.

Q. What are the risks to mother and baby due to diabetes?

Ans.

Risks to Mother

- An increase in incidence of pre-eclampsia, polyhydramnios, and operative delivery.
- Risk of developing diabetes later in life.
- Women with prior GDM are at greater risk for developing hypertension, hyperlipidemia, and electrocardiogram abnormalities. Mortality is also higher in these patients.

Risks to Baby

- Increased risk of perinatal mortality and morbidity.
- Increased risk of being large for gestational age, which increases the likelihood of birth trauma, induction of labor, and cesarean section. Shoulder dystocia and brachial plexus injury is common with macrosomia.
- *Risk of neonatal hypoglycemia:* When the umbilical cord is clamped, the fetal blood sugar level drops due to the continuing insulin secretion by the hyperplastic beta islet cells. This can be reduced by good maternal glycemic control during labor and birth (blood sugar <90 mg%) and early feeding of the baby.
- Neurodevelopment of the fetus may be hampered.
- Risk of the baby developing obesity and/or diabetes later in life.

Q. Why does the fetus develop macrosomia?

Ans. Maternal hyperglycemia leads to fetal hyperglycemia and fetal hyperinsulinemia which results in increased fetal growth particularly of fat and liver which are insulin sensitive.

Q. What specific antenatal care is advised?

Ans.

- Most patients in late pregnancy require relatively higher doses of insulin as they become increasingly insulin resistant during the third trimester of pregnancy.
- Women with diabetes should maintain fasting blood glucose between 70 and 110 mg% and 1-hour postprandial blood glucose below 140 mg% during pregnancy.

- Women with diabetes should be followed up for glycemic control every 1–2 weeks throughout pregnancy.
- Risk of hypoglycemia and hypoglycemia unawareness is high in pregnant patients on insulin, particularly in the first trimester, and close monitoring is required.
- During pregnancy, women who are suspected of having DKA should be admitted immediately in high dependency unit/monitored unit.
- Nephrology opinion should be sought in case of high serum creatinine (>1.35 mg%) or proteinuria.
- Babies should be kept in hospital for at least 24 hours to ensure adequate feeding and absence of hypoglycemia.
- Blood tests for polycythemia, hyperbilirubinemia, hypocalcemia, and hypomagnesemia should be carried out for babies with clinical signs.
- An echocardiogram is performed if clinical findings are suggestive of congenital heart disease or cardiomyopathy.
- Babies can be kept with their mothers unless there are abnormal clinical signs that warrant NICU (neonatal intensive care unit) admission.

Fetal Monitoring

- 2D echo of the fetal heart at 18–20 weeks
- Ultrasound monitoring of fetal growth and amniotic fluid volume every 4 weeks from 28 to 36 weeks.

Q. What are the implications of diabetes for labor and delivery?

Ans.

- Pregnant women with diabetes with a normal fetus can have either an elective vaginal birth through induction of labor or by elective cesarean section if indicated, after 38 weeks. Women with macrosomic babies may need a cesarean section.
- During labor and birth, blood glucose should be monitored hourly and maintained between 70 and 110 mg%.
- Women with type 1 diabetes may need IV dextrose and insulin infusion from the onset of labor to maintain blood sugar control.
- If general anesthesia is induced, blood glucose should be monitored every 30 minutes throughout the anesthetic period till the woman is fully conscious.
- In preterm labor, steroids can be given for fetal lung maturation but the insulin dose may need to be modified. Beta-mimetic drugs should be avoided for tocolysis.
- Women with diabetes should be advised to give birth in hospitals with round the clock support for advanced neonatal resuscitation.

Q. What special care is needed for babies born to diabetic mothers?

Ans.

- The major concern is hypoglycemia.
- Babies should be fed as soon as possible within 30 minutes and at 2–3 hours interval thereafter. The prefeed blood sugar should be above 36 mg%. Babies with low blood sugars or clinical signs of hypoglycemia should receive tube feeds or IV dextrose.
- Blood glucose testing should be carried out at 2–4 hours after birth.

Indications for NICU admission are:

- Hypoglycemia associated with abnormal clinical signs
- Respiratory distress
- Signs of cardiac decompensation due to congenital heart disease or cardiomyopathy
- Signs of neonatal encephalopathy
- Signs of polycythemia, likely to need partial exchange transfusion
- Need for IV fluids
- Need for tube feeding
- Jaundice requiring intense phototherapy and frequent monitoring of bilirubinemia
- Born before 34 weeks.

Q. How is blood sugar managed postpartum?

Ans. Women with gestational diabetes should discontinue hypoglycemic treatment immediately after birth. However, they need blood sugar examination at 6 weeks and annually thereafter.

Metformin and glibenclamide can be given to lactating women with pre-existing type 2 diabetes. First-generation sulfonylureas are secreted in milk and should be avoided.

Insulin dose should be reduced and blood sugar monitored frequently to achieve optimum control.

Women are at risk of hypoglycemia postnatally especially during breastfeeding and should be advised to have a meal or snack before or during feeds.

SUGGESTED READING

1. American Diabetes Association. 15. Diabetes Care in the Hospital: Standards of Medical Care in Diabetes-2020. *Diabetes Care*. 2020;43:S193–S202.
2. American Diabetes Association. Diagnosis and classification of diabetes mellitus. *Diabetes Care*. 2012;35:S64–71.
3. Association of British Clinical Diabetologists. (2013). Joint British Diabetes Societies Inpatient Care Group: The Management of Diabetic Ketoacidosis in Adults, 2nd edition. 2013. [online] Available from: <https://abcd.care/joint-british-diabetes-societies-jbds-inpatient-care-group> [Last accessed March, 2020].
4. Barker P, Creasey PE, Dhatariya K, et al. Peri-operative management of the surgical patient with diabetes. 2015

- guidelines, Association of Anaesthetists of Great Britain and Ireland. *Anaesthesia*. 2015;70:1427-40.
5. Coursin DB, Connery LE, Ketzler JT. Perioperative diabetic and hyperglycemic management issues. *Crit Care Med*. 2004;32:S116-25.
 6. Diabetes Times. (2012). The management of the hyperosmolar hyperglycaemic state (HHS) in adults with diabetes. [online] Available from: <https://diabetestimes.co.uk/nice-guidelines/the-management-of-the-hyperosmolar-hyperglycaemic-state-hhs-in-adults-with-diabetes/> [Last accessed March, 2020].
 7. Duggan EW, Carlson K, Umpierrez GE. Perioperative hyperglycemia management: an update. *Anesthesiology*. 2017;126:547-60.
 8. French G. Clinical Management of Diabetes Mellitus during Anaesthesia and Surgery. Update in Anaesthesia. 2000; 11:13.
 9. Kang H. Current therapeutic agents and anesthetic considerations for diabetes mellitus. *Korean J Anesthesiol*. 2012;63:195-202.
 10. Kitabchi AE, Umpierrez GE, Murphy MB, Kreisberg RA. Hyperglycemic crises in adult patients with diabetes: a consensus statement from the American Diabetes Association. *Diabetes Care*. 2006;29:2739-48.
 11. Levy N, Dhatariya K. Pre-operative optimisation of the surgical patient with diagnosed and undiagnosed diabetes: a practical review. *Anaesthesia*. 2019;74 Suppl. 1:58-66.
 12. McAnulty GR, Robertshaw HJ, Hall GM. Anaesthetic management of patients with diabetes mellitus. *Br J Anaesth*. 2000;85:80-90.
 13. National Institute for Health and Clinical Excellence. (2008). Diabetes in pregnancy. [online] Available from: https://www.ascalema.es/wp-content/uploads/2014/08/011KO_Diabetes-in-pregnancy-2008.pdf [Last accessed March, 2020].
 14. Robertshaw HJ, McAnulty GR, Hall GH. Strategies for managing the diabetic patient. *Best Pract Res Clin Anaesthesiol*. 2004;18:631-43.
 15. Schricker T, Carvalho G. Tight perioperative glycemic control. *Cardio Vas Anes*. 2005;19:684-8.
 16. Shetty SG, Crasto W, Jarvis J, Khunti K, Davies MJ. New insulins and newer insulin regimens: a review of their role in improving glycaemic control in patients with diabetes. *Postgrad Med J*. 2016;92:152-64.
 17. Shilling AM, Raphael J. Diabetes, hyperglycemia, and infections. *Best Pract Res Clin Anaesthesiol*. 2008;22: 519-35.
 18. Subramaniam RM, Suarez-Cuervo C, Wilson RF, Turban S, Zhang A, Sherrod C, et al. Effectiveness of prevention strategies for contrast-induced nephropathy: a systematic review and meta-analysis. *Ann Intern Med*. 2016;164(6): 406-16.
 19. Vinik AI, Maser RE, Mitchell BD, Freeman R. Diabetic autonomic neuropathy. *Diabetes Care*. 2003;26:1553-79.

Chronic Kidney Disease and Renal Transplant

Vandana Agarwal, Sohan Lal Solanki

A 66-year-old male hypertensive patient, who is currently on atenolol 50 mg and amlodipine 5 mg once daily, with a history of chronic kidney disease since 5 years posted for cholecystectomy. His serum creatinine is 5.7 mg%, serum potassium is 4 mEq/L, and the ECG shows T-wave inversion in inferior leads. How will you manage this patient?

Q. What are the stages of chronic kidney disease?

Ans. Chronic kidney disease (CKD) is a decline in the glomerular filtration rate (GFR) secondary to various diseases, such as diabetes, glomerulonephritis, and polycystic kidney disease.

Patients having GFR <60 mL/min/1.73 m² for >3 months are defined as having chronic renal disease. The stages of CKD described by the National Kidney Foundation Kidney Disease Outcomes Quality Initiative (KDOQI) Staging System for Chronic Kidney Disease are as follows:

- *Stage 1:* Kidney damage with normal or increased GFR (>90 mL/min/1.73 m²)
- *Stage 2:* Mild (GFR of 60–89 mL/min/1.73 m²)
- *Stage 3:* Moderate (GFR of 30–59 mL/min/1.73 m²)
- *Stage 4:* Severe (GFR of 15–29 mL/min/1.73 m²)
- *Stage 5:* End-stage renal disease (ESRD) (GFR of <15 mL/min/1.73 m²) or on dialysis.

Hemodialysis or peritoneal dialysis is usually initiated when the GFR decreases to <15 mL/min/1.73 m². Patients with ESRD are dependent on renal replacement therapy (RRT) for survival. Patients with CKD eventually need to undergo renal transplantation after initiation of hemodialysis or peritoneal dialysis. Following renal transplant, the 5-year survival rate is approximately 70%, whereas it is 30% in patients undergoing dialysis without a renal transplant.

Q. What pathophysiological changes occur in a patient with CKD and how can these affect the anesthetic management?

Ans. Patients with CKD may have many comorbid conditions and factors such as advanced age, hypertension, diabetes, lipid disorders, hyper-homocysteinemia,

abnormal calcium-phosphate metabolism, anemia, and uremic toxins, which increase their risk of perioperative morbidity. Patients with preexisting renal disease are more prone to the development of acute kidney injury (AKI) during their hospitalization. A small increase in serum creatinine can increase the risk of morbidity by as much as six times. The pathophysiological changes are discussed in the following text.

Fluid and Electrolyte Derangement

Sodium: CKD may be associated with sodium retention, sodium wasting, or abnormal sodium balance. This is further influenced by factors such as diuretic use and cardiac function. There is usually a certain degree of salt and water retention; however, the extracellular fluid volume remains isotonic. Patients with CKD may have impaired concentrating mechanisms and therefore they may not be able to compensate for extrarenal fluid losses, e.g., vomiting, diarrhea, or pyrexia. Thus, these losses can lead to hypovolemia and hypotension.

Potassium: Potassium secretion in the distal nephron may be affected and can lead to elevated potassium levels. However, this is dependent on factors such as diuretic use. Certain drugs such as β -blockers, potassium-sparing diuretics (spironolactone), angiotensin-converting enzyme (ACE) inhibitors or angiotensin antagonists, nonsteroidal anti-inflammatory agents, and nephrotoxins such as aminoglycosides and cyclosporins may cause acute hyperkalemia. Extracellular acidosis causes an exchange of intracellular potassium for extracellular hydrogen ions in an attempt to maintain electrical neutrality. In acute acidosis, the serum potassium will increase by 0.5 mEq/L for each

0.1 unit decrease in pH. Therefore, hypercarbia should be avoided during general anesthesia.

Magnesium: Magnesium clearance is affected and may lead to muscle weakness and potentiation of nondepolarizing muscle relaxants.

Acidosis: Chronic metabolic acidosis is a common feature of ESRD. The clearance of hydrogen ions is decreased because of the inability to secrete protons or buffers and regenerate bicarbonate. Reduced utilization of glutamine causes a decrease in ammonia production and secretion into the proximal tubule. Retention of organic anions causes a progressive increase in the anion gap and a further fall in plasma bicarbonate concentration.

Calcium, phosphate, parathormone, and renal osteodystrophy: Total plasma calcium concentration is reduced in CKD. Intestinal absorption of calcium decreases because of a fall in the production of vitamin D3. There is hyperphosphatemia as its excretion is impaired. Hyperphosphatemia causes calcium phosphate to be deposited in the soft tissues further lowering plasma calcium concentration. There is secondary hyperparathyroidism as a result of hypocalcemia and hyperphosphatemia, which increases the osteoclast and osteoblast activity, causing osteitis fibrosa cystica.

Hematological abnormalities:

- **Anemia:** CKD is associated with normochromic normocytic anemia. The causes of anemia are multifactorial:
 - Decreased renal erythropoietin (EPO) production leads to reduced stem cell transformation into erythrocytes
 - Reduced red cell life span due to uremia
 - Dietary deficiency of iron and folate
 - Chronic upper gastrointestinal tract losses and those from dialysis further compound the problem.

In CKD, this anemia is compensated by (1) shift of the oxyhemoglobin dissociation curve to the right because of increased 2, 3-diphosphoglycerate production, thus increasing oxygen delivery to the tissues; (2) increased cardiac output; and (3) decreased viscosity increasing microcirculation. These patients may be on recombinant EPO to improve the anemia associated with CKD. Target hemoglobin of 9.5 g% is maintained to improve exercise tolerance. EPO may aggravate hypertension and can lead to accelerated thrombosis of vascular access.

Coagulopathy: There is an increased tendency to bleed in the perioperative period. Standard tests of coagulation and platelet count are usually normal. The qualitative function

of platelets is impaired. The platelets have decreased adhesiveness and aggregation, probably due to impaired release of von Willebrand factor/factor VIII complex, which binds to and activates platelets. Also, there is increased release of β -thromboglobulin from the platelets and vascular production of prostacyclin (PGI_2) further contributing to coagulopathy. If perioperative bleeding occurs, platelet transfusion does not correct platelet dysfunction; however, dialysis may be helpful. Cryoprecipitate/DDAVP (increases release of von Willebrand factor) may be required to improve coagulation. DDAVP ($0.3 \mu\text{g/kg}$) is effective within 1–2 hours but has a short duration (6–8 hours); also tachyphylaxis may occur. The risks of bleeding complications should be considered when planning to use regional anesthetic techniques in CKD.

Cardiovascular Abnormalities

Systemic hypertension is the most common feature except in sodium-wasting nephropathies such as polycystic kidney disease or papillary necrosis. Sodium and water retention is the most frequent cause of hypertension and may be significantly improved by dialysis. Most of these patients are on antihypertensive drugs. Altered renin and angiotensin secretion may also contribute to hypertension.

Ischemic heart disease (IHD) is a frequent cause of mortality in patients with CKD. There is accelerated atherosclerosis secondary to impaired triglyceride clearance and lipoprotein lipase activity leading to impaired lipolysis. Hypertension may be a cause or effect of renal impairment.

Hypocalcemia and hyperphosphatemia lead to increased incidence of metastatic calcific valvular heart lesions. Patients on dialysis have a higher incidence of bacterial endocarditis. Before dialysis, hemorrhagic uremic pericarditis was often seen but is now uncommon and occurs in patients on an inadequate dialysis regimen. If untreated, it may progress to pericardial tamponade. Sudden death from acute cardiac arrhythmias may occur and is due to IHD and electrolyte abnormalities.

Pulmonary hypertension in patients posted for renal transplant predisposes the patient to an increased risk of postoperative hypotension and delayed graft functioning (need for hemodialysis within 7 days of surgery). Patients diagnosed with pulmonary hypertension should be treated before renal transplant surgery.

Pulmonary Abnormalities

Fluid overload, malnutrition, anemia, impaired humoral and cellular immune function, and decreased surfactant production predispose patients to atelectasis and infection.

Immune Function

The fistula and catheter site infections are common and wound healing is poor as a result of inhibition of cell-mediated immunity and humoral defense mechanisms.

The use of EPO and hepatitis B vaccine has led to a decrease in the incidence of viral hepatitis B. Universal precautions must be taken when caring for these patients.

Gastrointestinal Abnormalities

Uremia is a mucosal irritant and can lead to bleeding in the gastrointestinal tract. Patients with CKD have delayed gastric emptying leading to anorexia, nausea, and vomiting. They have an increased risk of aspiration. Coexisting diabetes mellitus further increases the risk of difficult intubation and aspiration as a result of autonomic gastroparesis. The rapid sequence induction technique needs to be balanced against the risks of difficult intubation. Succinylcholine may increase the plasma potassium concentration by approximately 0.5 mEq/L. Rapid sequence induction is usually done in patients who are full stomach or have symptoms of gastric reflux and have a low/normal level of serum potassium.

Neurological Abnormalities

Both the central nervous system (CNS) and the peripheral nervous system may be affected. CNS changes range from mild alterations in personality to asterixis, myoclonus, convulsions, and encephalopathy. Peripheral neuropathy occurs later as the CKD worsens. It is initially a distal "glove and stocking" sensory loss leading to motor involvement. Both dialysis and renal transplantation improve the neuropathy. The presence of a peripheral neuropathy implies autonomic neuropathy and this should alert the anesthetist, regarding delayed gastric emptying, postural hypotension, and silent myocardial ischemia. Two types of neurological disturbances are unique to patients on dialysis.

Dialysis dementia is a subacute, progressive, and potentially fatal condition. It occurs due to aluminum toxicity resulting from aluminum phosphate salts or aluminum in the dialysate. The incidence is low now as aluminum is removed from the dialysate. When it occurs, it is a multisystem disease that includes encephalopathy, osteomalacia, proximal myopathy, and anemia. The symptoms may range from dysarthria, apraxia, personality changes, psychosis, and myoclonus to convulsions and finally dementia progressing to death within 6 months.

Dialysis disequilibrium syndrome occurs in patients receiving hemodialysis. It is a self-limiting condition caused due to a reverse urea effect. Urea is cleared at a slower rate from the brain in comparison to that from the blood. This creates an osmotic gradient from the blood to the brain leading to transient cerebral edema, leading to headache,

nausea, vomiting, blurred vision, disorientation, delirium, hypertension, tremors, and convulsions.

Endocrine Disturbances

- Hyperparathyroidism as mentioned above
- Reduced/impaired production of EPO leads to anemia
- In CKD patients with diabetes, the requirement for exogenous insulin decreases probably due to reduced metabolism of insulin
- The temperature regulation is altered with a reduced basal metabolic rate predisposing to hypothermia.

To summarize, the main issues which need to be addressed prior to an elective procedure/surgery in a patient with CKD are:

- Progression of renal disease
- Prevention and management of fluid, electrolyte, and acid-base imbalances before and after surgery
- Need for emergency/maintenance dialysis in the perioperative period
- Prevention of further insults (iatrogenic), e.g., radio-contrast-induced acute renal failure
- High cardiac risk due to accelerated IHD
- Presence and evaluation of other comorbid conditions.

An early nephrology evaluation is mandatory to assess renal function and the need for RRT perioperatively. A multidisciplinary approach (anesthetist, cardiologist, nephrologist, surgeon, and physician) for perioperative management of these patients is essential for reducing cardiac and renal risks for the planned procedures.

Q. What are the causes of renal failure?

Ans. Diabetes mellitus is the most common cause of CKD in approximately 45% of the patients, followed by glomerulonephritis (20%), pyelonephritis (5%), and polycystic kidney disease (5%). Other causes are hypertension, systemic lupus erythematosus, etc.

Q. How does ESRD affect drug disposition in the body? Can you give examples of some commonly used anesthetic drugs?

Ans. Chronic kidney disease may alter both the pharmacokinetics and the pharmacodynamics of drugs. One must understand the effects of reduced clearance and accumulation of active metabolites along with the risks of worsening existing renal disease following drug administration. Dose adjustment is usually required if the GFR decreases below 50 mL/min/1.73 m². Metabolic acidosis due to the accumulation of organic acids may have significant effects on drug disposition and competition for an active transport process in the renal tubules.

Pharmacokinetics

Absorption: Absorption of drugs may be affected by delayed gastric emptying.

Distribution: The volume of distribution may be increased or decreased depending on the total body water, protein binding of drugs, and time since the last dialysis.

Protein binding:

- Protein binding of acidic drugs is decreased leading to increased concentration of active or free fraction of the drugs bound to albumin (e.g., salicylates, oral anticoagulants). This is due to:
 - CKD leading to hypoalbuminemia secondary to proteinuria
 - Accumulation of organic anions such as uric acid and lactic acid competes with the protein-binding site on albumin.
- Alpha-1 acid glycoprotein is bound to basic drugs (e.g., opioid analgesics, local anesthetics) and the levels are increased in CKD, thereby decreasing the unbound drug concentration.

Elimination: Drugs eliminated by the kidneys will have prolonged elimination half-lives, e.g., vecuronium.

Induction Agents

- *Propofol*: Its pharmacokinetics is not affected by CKD; however, careful administration titrated to clinical effect is suggested particularly in patients postdialysis as it can precipitate exaggerated hypotension.
- *Thiopentone*: In CKD, the volume of distribution is increased along with reduced plasma protein binding, thus increasing the active/free fraction of the drug requiring reduction in the dosage and rate of administration.

Inhalational Agents

- *Sevoflurane*: It reacts with CO₂ absorbents to form compound A which causes dose-related nephrotoxicity in rats. The metabolism of sevoflurane results in elevated fluoride levels (peak levels of 0.5 mmol/L). However, exposure to >4 minimum alveolar concentration (MAC) hours of sevoflurane was not associated with an increased risk of renal toxicity.
- *Desflurane*: It causes the least increase in fluoride level and therefore should be the first agent of choice if the vaporizer is available; however, it is expensive.
- *Isoflurane*: The increase in fluoride level is less compared to sevoflurane and is not associated with nephrotoxicity. It is readily available, is cost effective, and is therefore often the agent of choice in patients with CKD.

Neuromuscular Blockers (NMBs)

The initial dose (i.e., 3 × ED₉₅) required to produce neuromuscular block is larger in patients with CKD in comparison to normal patients due to increased volume

of distribution. However, maintenance of neuromuscular block during surgery requires reduced dose as metabolism and excretion of these drugs is affected in CKD except for atracurium and cisatracurium. One must avoid long-acting NMBs in patients with CKD, particularly the drugs excreted by the kidneys, and it is essential to monitor the neuromuscular block intraoperatively:

- *Succinylcholine*—when used for rapid sequence induction, it may increase the plasma potassium concentration by approximately 0.5 mEq/L; therefore, it should be avoided in patients with serum potassium ≥5.5 mEq/L. However, it can be safely used in patients with normal serum levels of potassium.
- *Atracurium*—undergoes Hoffman's elimination and hydrolysis by tissue esterases and does not depend on the kidneys for elimination. Laudanosine, an epileptogenic byproduct of atracurium metabolism, is known to accumulate in CKD and may be of concern in prolonged surgery.
- *Cisatracurium*—is superior to atracurium as it causes less histamine release, is more potent and produces less laudanosine, and has a shorter duration of action in comparison to atracurium.
- *Vecuronium and rocuronium*—predominantly excreted in the bile but up to 30% may undergo renal excretion. Vecuronium has an active metabolite 3-hydroxyvecuronium. CKD leads to reduction in clearance, prolonged elimination half-life, and duration of action.
- *Anticholinesterases*—neostigmine clearance is reduced and its half-life is prolonged in CKD. This may result in a prolonged parasympathomimetic activity, particularly when used in combination with atropine rather than with the longer acting glycopyrrolate.

Nonopioid Analgesics

- *Acetaminophen*: The use of acetaminophen is safe and does not require dose adjustment in the perioperative period.
- *Nonsteroidal anti-inflammatory drugs (NSAIDs)*: They are contraindicated in patients with renal failure including cyclooxygenase 2 (COX-2) inhibitors. They can cause hyperkalemia and hyponatremia and can exacerbate hypertension. They can cause bleeding in the perioperative period as a result of drug-induced platelet dysfunction, in addition to qualitative dysfunction caused by uremia. They inhibit the production of renal prostaglandins PGE₂ and PGI₂, which are responsible for autoregulating renal blood flow during hypovolemia and in the presence of vasoconstrictors, and thus can precipitate acute renal failure.

Opioid Analgesics

- *Morphine* is predominantly metabolized in the liver to morphine-3-glucuronide (M3G) and about 5% to morphine-6-glucuronide (M6G). M3G antagonizes the analgesic effect of morphine, causes irritability, and lowers the seizure threshold. M6G has potent analgesic properties that are responsible for the delayed onset of sedation and respiratory depression. M6G gets eliminated by the kidneys and so in patients with CKD, it can accumulate and the half-life may prolong from 2 to 27 hours. Oral morphine undergoes extensive first-pass metabolism in the liver and therefore the concentration of active metabolite is greater than the equianalgesic parenteral dose. In patients with CKD, the dose and frequency of morphine should be reduced and the patient should be monitored for delayed respiratory depression postoperatively. In patients on dialysis, a significant fraction of morphine is removed.
- *Fentanyl* undergoes extensive hepatic metabolism with no active metabolites. Approximately 7% is excreted unchanged in the urine. However, the clearance is decreased in severe uremia.
- *Alfentanil* clearance is not affected by renal failure and it can be administered safely.
- *Remifentanyl* undergoes metabolism by nonspecific blood and tissue esterases and it does not accumulate in patients with ESRD. Its primary metabolite does accumulate but the effect is clinically insignificant as it has low potency, and pharmacokinetic simulations suggest that it does not reach clinically significant concentrations even after 24 hours of infusion.
- *Tramadol* is metabolized to O-desmethyltramadol, an active metabolite, which is excreted by the kidneys. In addition, 30% of tramadol is excreted unchanged by the kidneys. Also, tramadol may be epileptogenic in these patients as uremia further lowers the seizure threshold. Extended-release tramadol should be avoided in patients with CKD. The dosing interval of tramadol (regular release) may need to be increased to every 12 hours in patients with a creatinine clearance <30 mL/min. Tramadol is removed by hemodialysis.
- *Meperidine (pethidine)* should be avoided in patients with CKD as it is metabolized to normeperidine. Normeperidine is associated with seizures, myoclonus, and altered mental state and depends on the renal function for elimination.
- *Codeine and dihydrocodeine* are also best avoided as their elimination half-life is significantly prolonged, and conventional doses may result in central nervous system depression.
- *Local anesthetics* have a shorter duration of action due to altered protein binding. The maximum doses of local

anesthetics should be reduced by about 25% because of altered protein binding and a lower seizure threshold.

Q. How will you conduct the anesthesia for this patient posted for nontransplant surgery such as appendicectomy?

Ans. The main anesthetic goal is to maintain renal perfusion and prevent harm to the already compromised renal function by avoiding hypoxia, hypovolemia, and hypotension.

Preoperative Evaluation

History: Establish the cause of renal failure and duration of treatment. Also, discuss with the patient the need for dialysis postoperatively. Enquire about fluid restriction if any and daily urine output. Elicit a history of comorbidities (hypertension, diabetes, IHD, connective tissue disorders) and whether controlled and on what treatment (dose, frequency).

Enquire about exercise tolerance, anemia, left ventricular failure (LVF), electrolyte disturbance, medications (antihypertensives, diuretics, sodium bicarbonate, vitamin D3, resonium), and symptoms of gastroesophageal reflux. Seek nephrology opinion regarding the need for dialysis in the postoperative period.

Examination: Apart from the general examination, measure the patient's blood pressure (BP) in standing and in sitting positions which may suggest autonomic neuropathy. They may have flow murmur secondary to anemia and pericardial rub due to uremic pericarditis. Look for ankle or sacral edema which may indicate either right ventricular failure or hypoproteinemia or both. Patients who are fluid overloaded may have crepitations.

Investigations

- *Full blood count:* Normochromic, normocytic anemia, and infection are likely.
- *Clotting studies:* They are required if the uremia is severe.
- *Renal function tests* [blood urea nitrogen (BUN), serum creatinine, electrolytes] are mandatory prior to surgery. Creatinine is a good measure of the GFR. Usually, these patients have their renal functions tested periodically and looking at the trend will help in eliciting the progression of the disease. Also, the creatinine clearance is regularly measured and knowledge of the same will help in dose adjustments of various medications in the postoperative period.
- *Electrocardiogram (ECG):* Look for ischemia, arrhythmia, left ventricular hypertrophy (LVH), conduction blocks, or hyperkalemia.
- *Chest radiograph:* Pleural effusions, cardiomegaly, pulmonary edema.

- **Arterial blood gas (ABG):** To evaluate the acid-base status.
- **Liver function test (LFT):** If a major surgery is planned; as a baseline value.

Preoperative Optimization/Preparation

For elective surgery, it is prudent to optimize BP and serum potassium levels. Avoid unnecessary blood transfusion because of anemia to avoid sensitization for future transplantation. The antihypertensive should be continued perioperatively. Metoclopramide and H₂ receptor antagonists should be administered if the patient has gastroesophageal reflux.

Induction of Anesthesia

- Preoxygenate.
- Administer induction drugs slowly to minimize hemodynamic disturbances. If hypotension occurs despite above, vasopressors can be given titrated to mean arterial pressure (MAP).
- If rapid sequence induction is necessary (inadequately fasted, delayed gastric emptying, history of gastroesophageal reflux), suxamethonium can be used if the serum potassium is <5.5 mEq/L; alternatively, modified rapid sequence induction can be done with rocuronium (if one does not anticipate difficult airway). If difficult airway is anticipated, inhalational induction is a safer option.
- If there is any doubt regarding airway adequacy, always intubate.
- Regional anesthesia can be administered after weighing the risks and benefits. The concern with epidural anesthesia is platelet dysfunction, increasing the risk of epidural hematoma.

Maintenance of Anesthesia

- Nitrous oxide can be administered safely as it does not affect the renal function.
- Isoflurane is the inhalational agent of choice as only 0.2% undergoes metabolism and produces low levels of fluoride ions.
- Ventilation should be controlled for long procedures. If the patient has been on conservative management for renal failure, compensatory changes in the PaCO₂, pH, oxyhemoglobin dissociation curve, and cardiac output would have occurred to maximize oxygen delivery to the tissues; therefore, ventilation should be adjusted accordingly.
- Cisatracurium/Atracurium is preferable as it undergoes Hoffman's elimination. Vecuronium and rocuronium can be used but smaller top-up doses are required less frequently. The neuromuscular blockade should be monitored using a nerve stimulator and top-up doses administered accordingly.

- Fentanyl can be administered safely; however, its half-life may be prolonged particularly if used as an infusion. Morphine can be used with care as clearance is reduced leading to accumulation. If morphine is administered, the patient should be monitored for respiratory depression in the postoperative period. It should never be used as a continuous infusion.

- **Monitoring:** Apart from an electrocardiogram, non-invasive BP (on the nonfistula arm), pulse oximetry, end-tidal CO₂ and temperature monitoring, and arterial BP monitoring may be used in selected patients with uncontrolled hypertension despite taking multiple antihypertensive drugs or in patients with limited cardiac reserve. Central venous cannulation is required for continuous central venous pressure (CVP) monitoring intraoperatively and postoperatively to maintain high intravascular volume because the transplanted kidney is denervated and is pressure- and volume-dependent for initial proper functioning. Dynamic indices of fluid responsiveness such as pulse pressure variation, stroke volume variation, and esophageal Doppler-guided fluid therapy can be used in renal transplant recipients.

Neuromuscular monitoring should be done to guide the intraoperative neuromuscular blockade and for tracheal extubation after the surgery because uremia may prolong the neuromuscular blockade.

- A urinary catheter should be passed to monitor urine output hourly and should be maintained at 0.5–1 mL/kg/h. If the urine output is low despite good hydration and BP, mannitol should be administered as a first-line treatment followed by furosemide.
- Intravenous (IV) fluids should be administered cautiously, as these patients may be fluid overloaded, and if required central venous monitoring may be done to guide volume replacement.
- Forced air warmer and fluid warmer should be used mandatorily to maintain normothermia as hypothermia (< 35°C) can potentiate the action of most of the drugs, thus delaying recovery from anesthesia.
- **Colloids and IV fluids:** Starches should be avoided in these patients as they can accumulate and worsen the renal impairment. Gelatine is safe. Ringer's lactate can be used if serum potassium is within the normal limits. Overtransfusion of blood to correct anemia should be avoided as an increase in hematocrit can decrease renal perfusion and further compromise the renal function.
- NSAIDs are contraindicated in these patients; paracetamol can be administered safely.

Reversal of Neuromuscular Block

Neuromuscular blockade can be reversed with neostigmine. Recurarization is a potential problem that can occur in the postoperative period and the recovery staff should be warned about the same. If there are any concerns about inadequate reversal, it is safer to ventilate the patient for short term till complete neuromuscular recovery occurs.

Postoperative Care

Oxygen should be administered. ECG, BP, and respiration and pulse oximetry should be monitored closely in the postoperative period. Analgesia should be administered. Early mobilization and physiotherapy are important. Dosages of medications should be administered as per creatinine clearance if known; alternatively creatinine clearance can be estimated by the following formula:

Cockcroft-Gault equation (eCCr = estimated creatinine clearance)

$$eCCr = \frac{(140 - \text{Age}) \times \text{Mass (in kilograms)} \times (0.85 \text{ if female})}{72 \times \text{Serum creatinine (in mg/dL)}}$$

After minor surgery, the patient should return to his/her preoperative regimen as soon as possible. After a major surgery, some patients may require dialysis for some time in the postoperative period.

Q. What is an arteriovenous fistula (AVF)? How will you anesthetize this patient for AVF surgery?

Ans. Patients with CKD may have to undergo AVF surgery, a couple of months preceding dialysis. A fistula is surgically created between the radial artery and the cephalic vein. Other sites include brachial and basilic veins. The purpose of the AVF is to improve venous access for subsequent dialysis, i.e., dilates the vein sufficiently to allow easier access. This takes about 4–8 weeks to establish. Complications include infection, stenosis, thrombosis, aneurysm, and limb ischemia. This procedure may take a considerable amount of time.

Various options available are general anesthesia, local infiltration anesthesia, and regional anesthesia with brachial plexus block.

- *General anesthesia (GA)* is not without complications in view of significant comorbidities but may have to be administered if the patient refuses or is not cooperating for regional anesthesia. If GA has to be administered, the same considerations apply as for a CKD patient undergoing non-transplant surgery.
- *Local infiltration anesthesia* is not ideal for an extensive procedure. Patient cooperation is very essential. Repeated injections may be required; this can increase the chances of systemic local anesthetic toxicity as mentioned earlier.

- *Regional anesthesia (RA) with a brachial plexus block* is an effective and preferred alternative to GA and infiltration anesthesia. RA has various advantages such as vasodilatation which helps in selecting the site for venous access, avoidance of hemodynamic instability and stress response with general anesthesia, excellent postoperative analgesia, and motor block. It can be administered in a patient with cardiorespiratory compromise. When conducting the case under RA, the maximum dose of the local anesthetic drug should be reduced by 25% because of its altered pharmacokinetics in CKD and lower seizure threshold, which predisposes these patients to local anesthetic toxicity.

Q. What will be your anesthetic plan if this patient was dialysis dependent?

Ans. Dialysis (hemodialysis/peritoneal dialysis) is usually initiated if the creatinine clearance is <15 mL/min. An AVF is created usually on the nondominant hand. It is important to establish the weekly frequency of dialysis.

Essentially, the consideration and preparation are the same as mentioned earlier (nondialyzed CKD patient for nontransplant surgery). Anesthetic considerations related to dialysis patients are mentioned below.

Preoperative Evaluation

- Discuss with the nephrologists regarding fluid management of the patient. It is important to coordinate the timing of dialysis and surgery. The dialysis should finish 4–6 hours preoperatively to allow time for a fluid shift; heparin is to be eliminated. After dialysis, full blood count, urea, creatinine, electrolytes, clotting studies, and patient's weight should be measured.
- The AVF site should be noted and that limb must be carefully protected during the surgery. The BP cuff should never be placed on the same arm; also an IV line should be secured on the other hand or leg. Regional anesthesia can be safely administered if the clotting study is normal.
- These patients are very sensitive to induction agents and volatile agents, particularly soon after dialysis. Therefore, titrated and slow administration of induction agents will help in minimizing hemodynamic disturbances.
- Overtransfusion is not a concern as the GFR does not need to be maximized. The only concern is sensitization for future transplantation; usually, washed red cells should be transfused if facilities are available.

Q. How will you manage acute hyperkalemia?

Ans.

- ECG should be monitored, if there is wide QRS complex and tall peaked T wave, it should be treated urgently.

- The first-line treatment is calcium chloride/gluconate (to stabilize the myocardium) and sodium bicarbonate (to reduce the acidosis). The two drugs should never be given in the same line as calcium carbonate may be precipitated.
- This should be followed by glucose insulin (a temporary measure to drive the potassium intracellularly).
- The excess potassium can be removed from the body by ion exchange resins and dialysis.

Q. What will be your anesthetic plan for a CKD patient undergoing renal transplant surgery?

Ans. The main anesthetic goal is to maintain renal perfusion and prevent harm to the donated kidney by avoiding hypoxia, hypovolemia, hypotension, and nephrotoxic drugs. The shorter the duration of RRT, the better the outcome following renal transplant. These patients undergo dialysis a day before the surgery to optimize volume (within 0.5 kg of ideal body weight), acid-base status, and potassium levels. Preoperative dialysis improves graft function and decreases mortality. The anesthetic considerations are essentially the same as mentioned earlier. Certain specific concerns related to transplant surgery are mentioned below:

- These patients undergo a comprehensive medical evaluation for transplant. Sometimes, the transplant surgery may be done on an emergency basis, particularly in those patients undergoing a cadaveric renal transplant. Preoperative history and investigations should be reviewed prior to surgery. Steroids and immunosuppressive agents are administered preoperatively.
- Antihypertensives should be continued perioperatively.
- The AV fistula should be protected; central venous and arterial cannulas should be secured on other extremities. The fistula site should be padded and palpated frequently to check patency. If the fistula patency is lost, the surgeon should be informed immediately.
- Epidural can be safely placed in patient with normal platelet count and who is undergoing regular hemodialysis because toxins (phenol, phenolic acid and guanine-succinate) that get accumulated and cause platelet dysfunctions are cleared by regular hemodialysis). Preference for epidural placement varies amongst renal units. Some units do not prefer epidural analgesia because of platelet dysfunction and possible neuraxial complications and infection.
- Blood should be made available and transfused if required.
- IV methylprednisolone is usually given midway of the surgery.
- Invasive monitoring (CVP and arterial) is established to guide fluid replacement.
- Post-transplantation after the renal clamps are removed, it is important to maintain perfusion to the transplanted kidney by restoration of circulatory volume (target CVP ≥ 10 –12 mm Hg) and avoiding hypotension.
- The surgery may take hours and involve major fluid shifts. Efforts should be taken to maintain normothermia.
- Anesthetists may be asked to administer diuretics to promote the function of the kidney and increase urine production. Mannitol and furosemide may be used to stimulate urine production and to restore function in the transplanted kidney after reperfusion.
- Mannitol 20–50 g (100–250 mL of 20% mannitol) is used immediately before opening the vascular anastomosis and reduces the incidence of acute renal failure. It expands the intravascular volume, increasing the tubular flow by preventing water absorption in the proximal convoluted tubule. It also enhances the release of prostaglandins in the kidney and may act as a free-radical scavenger.
- Furosemide 200–500 mg is commonly given while the vascular anastomosis is created, to stimulate diuresis. Its role in preventing renal failure is uncertain; the only indication for loop diuretics is fluid overload. Mannitol is always preferred over furosemide.
- Corticosteroids, used as a part of the immune suppression protocol, may also improve renal function.
- Hyperkalemia should be treated with insulin, glucose, hyperventilation, and calcium if significant. Once the renal function is restored following transplant, hyperkalemia is controlled easily. If the kidney function is not restored, some patients may need dialysis in the postoperative period for hyperkalemia and fluid overload.
- Intraoperative hypertension may occur because of hypervolemia (preoperative dialysis is essential for hypervolemia). Sympathetic stimulation secondary to surgical stress and pain should be treated by supplementing analgesia, deepening the anesthetic depth, and using short-acting antihypertensive drugs. Sublingual nifedipine should be avoided as it causes a precipitous fall in BP, which can increase the perioperative morbidity.
- Hypotension occurs in the post-transplant period which can affect graft function. Therefore, BP should be maintained perioperatively by restoring the intravascular volume and using vasopressors if required.
- Regional anesthesia/analgesia can be administered after weighing the risks and the benefits. Concerns with epidural anesthesia are effect of residual heparin after preoperative dialysis and platelet dysfunction, theoretically increasing the risk of epidural hematoma. Also, hemodynamic instability can adversely affect graft function.

Postoperative care: Most patients can be extubated at the end of the surgery. Oxygen should be supplemented and all invasive monitoring should be continued. Urine output should be monitored closely. Maintaining renal perfusion is the main goal in the postoperative period. The CVP should be maintained between 10 and 12 mm Hg. Alternatively, advanced cardiac output monitoring can be instituted to guide fluid therapy. 5% dextrose, 0.45% normal saline, or 0.9% normal saline is the recommended maintenance fluid at 30 mL/h plus previous hour's urine output. Oral hydration may be started once the patient is awake post-transplant. Immunosuppressive drugs are continued in the postoperative period. The commonly used drugs are cyclosporine A, azathioprine, monoclonal antibodies, and steroids. Newer drugs such as tacrolimus and mycophenolate mofetil may replace cyclosporine A and azathioprine, respectively, in some immunosuppression protocols. The drug levels of cyclosporine A or tacrolimus must be maintained within the therapeutic range; therefore, daily drug level monitoring is done in the perioperative period.

Postoperative analgesia: Patient-controlled analgesia (PCA) with morphine or fentanyl can be used. There is no evidence to support one opioid over the other in post-transplant patients. Accumulation of metabolites may act as negative feedback and limit PCA use. A bolus dose of opioid must be reduced (0.5 mg bolus with a 10–15-minute lockout period). The patient should be closely monitored for respiratory depression. Epidural analgesia can be used, but hypotension should be avoided. NSAIDs are contraindicated.

Q. What is cold and warm ischemia time?

Ans. Ischemia time starts with clamping of the renal vessels in the donor and ends with vascular anastomosis in the recipient. The shorter the duration of ischemia time, the better the organ preservation. Warm ischemia time begins with the clamping of the renal vessels in the donor. It is interrupted by cold preservation solution and resumes again when the kidney is placed in the recipient and ends with the vascular anastomosis.

Cold ischemia: The kidney is preserved by storing at 4°C usually for <24 hours.

Q. What solutions are used for organ preservation?

Ans. There are three types of organ preservation solutions used for kidney preservation after procurement from a donor:

1. **EuroCollins:** This is a simple and cheap intracellular-like (high potassium and low sodium) preservation solution. In this, phosphates are used as buffering agents and

glucose is used as a source of energy. Its use is declining in western countries because of increased reports of delayed graft function if used.

2. **University of Wisconsin (UW) solution:** Currently, this solution is most commonly used. It consists of metabolically inert substances such as lactobionate and raffinose served as an osmotic agent. Hydroxyethyl starch (HES) is used as colloid and it also has scavengers such as glutathione and allopurinol and for energy source adenosine is present. This is an ideal and gold standard solution for organ preservation for kidney, liver, and pancreas.
3. **Histidine-tryptophan-ketoglutarate (HTK) solution:** This was originally introduced as a cardioplegic solution for open-heart surgery and later used for other organ preservation. It consists of strong buffer histidine and two amino acids: tryptophan, which acts as a membrane stabilizer and antioxidant, and ketoglutarate, which acts as a substrate for anaerobic metabolism during preservation. It has low viscosity and to achieve complete tissue equilibrium, high volume (approximately 15 L) is required. HTK solution is considered superior when the kidney is harvested from a cadaveric donor.

Q. What is a kidney cocktail?

Ans. The kidney cocktail is infused during the vascular anastomosis in the recipient (only for cadaveric kidneys). It consists of 600 mL of 0.45% dextrose in 0.45% normal saline, 37.5 g of albumin, 80 mg of furosemide, and 37.5 g of mannitol. (Some renal transplant units use the kidney cocktail; it may not be used in all units).

Q. What postoperative complications can occur following renal transplant surgery?

Ans. The most common complications occurring in up to 70% of patients are AKI and delayed graft function, requiring dialysis. Delayed graft function usually occurs because of acute tubular necrosis (ATN) either due to ischemic damage prior to transplantation or due to hypoperfusion before and after transplant.

Q. How will you manage postoperative analgesia in a patient who has undergone renal transplant?

Ans. Postoperative analgesia in renal transplant recipients can be managed by epidural analgesia with local anesthetic solution.

In the absence of epidural analgesia, fentanyl-based patient-controlled analgesia, along with intravenous followed by oral paracetamol can be administered.

Weaker opioids like intravenous/oral Tramadol or Tapentadol may be used for mild to moderate acute pain in the postoperative period. Tapentadol has a dual mechanism of analgesia, it is a μ -receptor agonist and

also inhibits reuptake of norepinephrine and serotonin in the neurons. It has an improved side effect profile compared to opioids and non-steroidal anti-inflammatory drugs (NSAID's). NSAID's and extended release tramadol should be avoided. Paracetamol is safe to administer.

Preoperative (post induction of anesthesia) or after completion of surgery transverse abdominal plane block may be used. It has an opioid sparing effect in renal transplant recipients. Needle and catheter should be placed cautiously, if inserted after surgery especially on the side of transplanted kidney.

Q. What are the anesthetic considerations if this patient, now a postrenal transplant, is posted for nontransplant surgery?

Ans. The 1-year survival rate following a transplant is improving. A number of these patients present for elective and emergency surgery. Therefore, anesthetists and surgeons may be required to manage these patients in hospitals which are not involved in transplant procedures. The main considerations for post-transplants are:

- Physiological and pharmacological problems of the grafted kidney
- Side effects of immunosuppressive drugs
- Risks of graft rejection
- Infection.

Q. Discuss pharmacological problems seen in post-transplant patients.

Ans. The usual immunosuppressive regimen includes triple therapy, consisting of a calcineurin inhibitor (cyclosporine or tacrolimus), an antiproliferative agent (azathioprine or mycophenolate mofetil), and a corticosteroid. Newer regimens include polyclonal/monoclonal antibodies and attempt to eliminate calcineurin inhibitors and corticosteroids:

- *Calcineurin inhibitors* form the mainstay of immunosuppression therapy.
- *Cyclosporine*: It may cause hypertension, hyperlipidemia, hyperkalemia, gum hypertrophy, and nephrotoxicity with renal fibrosis. They lower the seizure threshold so hyperventilation should be avoided. Hyperkalemia and hypomagnesemia are frequently seen. It potentiates neuromuscular block and may be associated with postoperative respiratory failure.
- *Tacrolimus*: It is similar to cyclosporine but may have a better cardiovascular risk profile, with less hypertension and hyperlipidemia. Although it is also nephrotoxic, it is associated with improved long-term post-transplant renal function. Important adverse effects include disturbed glucose metabolism and diabetes mellitus, tremor, diarrhea, neurotoxicity, and nephrotoxicity.

Antiproliferative Agents

- *Mycophenolate mofetil* has replaced azathioprine. It acts by inhibiting de novo purine synthesis in lymphocytes. It is not nephrotoxic. The oral bioavailability is 94%.
- *Azathioprine* causes bone marrow suppression and can lead to pancytopenia requiring dosage adjustments. It may transiently antagonize neuromuscular block which is clinically insignificant.
- *Steroids* are the mainstay of post-transplant immunosuppression and continue to be used for the prevention and treatment of graft rejection. Patients on long-term use may also have steroid-related side effects.

Conduct of Anesthesia

The main anesthetic goal is to maintain renal perfusion with the least alteration in the immunosuppressive regimen. Other considerations are similar to any CKD patient undergoing surgery. Important anesthetic considerations in post-transplant recipients are mentioned in this section.

Preoperative Evaluation

History: One should elicit information regarding the:

- Functioning of graft and any evidence of rejection if the function is abnormal. The presence of rejection should be ruled out preoperatively. The presence of uremia, hypertension, and proteinuria may indicate chronic graft rejection. Some postrenal transplant patients may require dialysis; all the necessary evaluation should be done as described earlier.
- Rule out any evidence of infection as immunosuppressive drugs predispose these patients to bacterial, viral, fungal, or protozoal infections. They do not present with typical signs and symptoms of intra-abdominal sepsis such as fever, raised white cell count, and physical signs of peritonitis.

Other organ functions may be affected secondary to side effects of immunosuppressive therapy. Upper GI bleeding may occur probably secondary to acid peptic disease, gastritis, or cytomegalovirus gastroenteritis. Hepatobiliary and pancreatic diseases are common after transplantation.

All baseline investigations should be done including liver function tests as they may be deranged secondary to immunosuppressive drugs.

Induction of Anesthesia

- The choice of perioperative monitoring is determined by the nature of surgery.
- Nasal endotracheal intubation should be avoided to prevent infection caused by nasal flora. Laryngeal mask airway can be used safely.
- Regional anesthesia can be administered if the clotting studies and platelet count are normal. Azathioprine or antithymocyte globulin can cause thrombocytopenia,

which increases the bleeding and hematoma risks associated with central neural blockade.

- **Drugs**—postrenal transplant patients may have a normal serum creatinine value; however, their GFR is low in comparison to normal individuals so the renal elimination of drugs may be prolonged and drugs should be administered cautiously:
 - NSAIDs are contraindicated and can augment nephrotoxicity of cyclosporine.
 - Drug interactions—cyclosporine A interacts with nondepolarizing NMBs potentiating their action; thus, decreasing the dose and frequency of top-up doses is advised. Azathioprine antagonizes neuromuscular blocking drugs.
 - The drug levels of cyclosporine A or tacrolimus must be maintained within the therapeutic range; therefore, daily drug level monitoring is done in the perioperative period.
 - Oral cyclosporine should be administered 4–7 hours prior to surgery to maintain therapeutic levels.
 - The immunosuppressive agent should not be stopped perioperatively; the dose may have to be altered if the route of administration is to be changed. Oral prednisone can be converted to IV methylprednisolone. Stress doses of steroids should be given at induction depending on the type of surgery. Patients should be started on the oral dose as soon as possible; alternatively, IV supplementation should be done.
 - For azathioprine, the oral and IV doses are equivalent.
- Severe perioperative airway obstruction may occur because of the underlying post-transplant lymphoproliferative disease.

Q. What are your perioperative concerns for laparoscopic/robotic-assisted surgery in postrenal transplant patients?

Ans. The laparoscopic and robotic-assisted approaches have emerged as minimally invasive alternatives to open surgery, with several cases reported using a transperitoneal approach. The general benefits of laparoscopic surgery include less blood loss, quick recovery, cosmetically better, low postoperative morbidity, and short hospital stay. Perioperative injury and loss of renal allograft because of laparoscopic ports are major concerns. To prevent this, alternative port types and sites are chosen. The majority of postrenal transplant patients continue to be hypertensive and the immunosuppressive agents cause hypertension as a side effect. The perioperative goal for MAP should be higher than that of a normal patient. It is imperative to maintain the MAP to maintain renal perfusion, and intra-abdominal pressure should be taken into consideration for the same. Most of the

lymphoceles after renal transplant can successfully be treated by laparoscopy under general anesthesia.

Q. Postrenal transplant patients are on steroids. How will you supplement steroids perioperatively?

Ans. All patients who are taking <10 mg prednisolone have been shown to have a clinically normal response to hypothalamic-pituitary-adrenal (HPA) testing. These patients do not need additional steroid cover other than their usual steroid dose. This should be taken preoperatively and continued as soon as oral intake is possible.

Patients who are on long-term steroid supplementation equivalent to >10 mg prednisolone daily (or who have received such a dose within the last 3 months) should receive a physiological replacement regimen as mentioned in **Table 1**. Alternatively, adrenal suppression should be excluded by preoperative biochemical testing.

TABLE 1: Perioperative steroid treatment regimen.

Patients who have received a regular daily dose of >10 mg prednisolone or equivalent in the last 3 months

Minor surgery (hernias, hand surgery)	25 mg hydrocortisone at induction
Moderate surgery (hysterectomy)	Usual preoperative steroids + 25 mg hydrocortisone at induction + 100 mg hydrocortisone/day
Major surgery (major trauma, prolonged surgery, or surgery where there is delayed oral intake)	Usual preoperative steroids + 25 mg hydrocortisone at induction + 100 mg hydrocortisone/day for 2–3 days. Resume normal oral therapy when the gastrointestinal function has returned

All other patients—no additional steroids required

Q. What are the measures to prevent postoperative acute kidney injury (AKI) in patients with normal and impaired renal function?

Ans. Renal impairment following surgery is most often associated with multiorgan dysfunction, increasing postoperative morbidity and length of stay in the intensive care unit (ICU) and hospital and later may progress to CKD and dialysis-dependent ESRD.

Prevention Strategies

- Nonpharmacological measures such as:
 - *Intravascular volume expansion:*
 - Both crystalloids and certain colloids can be used in patients with renal impairment.
 - Albumin and gelatin are safe in patients with normal renal function.
 - The safety of HES solutions in patients with renal failure is not established. When HES was compared to Ringer's lactate in critically ill patients with severe sepsis, it was associated with a higher incidence of bleeding and AKI.

- Postsurgical patients who receive IV contrast will benefit from the use of isotonic IV fluids and nonionic, iso-osmolar contrast in the lowest possible dose/volume.
- *Maintenance of renal blood flow and renal perfusion pressure:*
 - The first step is to restore the circulating volume and correct hypovolemia.
 - Vasopressor and inotropes should be subsequently used for the management of systemic arterial hypotension and low cardiac output, respectively. Norepinephrine is an excellent first-line vasopressor agent. At present, there is no confirmatory evidence to prove that it decreases renal, hepatic, or gastrointestinal blood flow when used to treat arterial hypotension.
 - A target mean arterial pressure of 65–75 mm Hg is used in clinical practice; however, in hypertensive patients, it should be higher depending on their BP control as the autoregulation shifts to the right.
- *Avoidance of nephrotoxic agents:*
 - The use of once-daily aminoglycoside dosing and the use of liposomal amphotericin B have decreased the risk of nephrotoxicity associated with these drugs.
- *Early and appropriate management of postoperative complications:*
 - Early diagnosis and treatment of postoperative complications such as acute cardiac dysfunction, hemorrhage, sepsis, and intra-abdominal hypertension/compartiment syndrome are essential to prevent the development of AKI.
 - Abdominal compartment syndrome caused by intra-abdominal hypertension decreases renal perfusion and may precipitate ischemic ATN. A high index of suspicion and early recognition by intravesical pressure measurement help in diagnosing this condition. If detected, decompressive laparotomy is usually needed.
- *Pharmacological measures:* Various drugs have been used as protective agents for preventing perioperative renal dysfunction such as dopamine agonists (dopamine, dopexamine, fenoldopam), diuretics (loop and osmotic diuretics), natriuretic peptides, and N-acetyl cysteine. Most of the studies were done in patients who underwent cardiac surgery or in critically ill patients with renal dysfunction.
 - *Dopamine:* A large multicenter trial demonstrated that it does not prevent AKI. It neither avoids the need for RRT nor decreases the mortality. Perioperatively, it increases the urine output but has no effect on the outcome. Also, systematic reviews

have concluded that there is no role of low-dose (renal dose) dopamine for renal protection.

- *Fenoldopam:* It does not prevent radiocontrast-induced nephropathy in patients with preexisting renal impairment, has no effect on the outcome in critically ill ICU patients with early acute renal impairment, and is not renoprotective perioperatively in high-risk cardiac surgical patients.
- *Diuretics:* Both the loop and the osmotic diuretics do not demonstrate significant renal protection in the perioperative setting; however, there is limited evidence and a future trial has been proposed.
- *N-acetylcysteine (NAC):* There is evidence to support its prophylactic use as an antioxidant along with isotonic IV fluids in the prevention of radiocontrast-induced nephropathy. Recent trials in perioperative and critically ill patients do not demonstrate the renoprotective effect.

A Cochrane review evaluating available evidence regarding pharmacological interventions (including the use of dopamine and its analogs, diuretics, calcium channel blockers, ACE inhibitors, or hydration fluids) for perioperative renal protection suggested that there is no evidence that these drugs are beneficial. Also, there was no difference in morbidity or mortality.

Patients at risk:

- Undergoing cardiac/vascular surgery/major surgery
- Elderly
- Preexisting renal dysfunction
- Persistent hemodynamic instability leading to renal hypoperfusion
- Perioperative use of nephrotoxic drugs
- Postrenal transplant.

Q. What are RIFLE, AKIN, and KDIGO criteria for grading renal dysfunction? Compare and contrast the above criteria.

Ans. Various grading systems can be used to classify AKI. They are RIFLE (R, risk; I, injury; F, failure) and two outcome variables (L, loss; E, end-stage), AKIN (Acute Kidney Injury Network) and KDIGO (Kidney Disease Improving Global Outcomes) and urine output (**Table 2**). An international interdisciplinary collaborative group, the acute dialysis quality initiative (ADQI), was created in 2002 for the development of guidelines for the prevention and treatment of AKI. Recently, the group has created a standard grading system for acute renal dysfunction. In 2007, the Acute Kidney Injury Network (AKIN) was formed and coined the term AKI. The main purpose was to represent the full spectrum of AKI from mild to severe. Severe AKI was associated with an increased likelihood of unfavorable outcomes, i.e., loss of function and ESRD.

TABLE 2: RIFLE, AKIN, and KDIGO grading systems.

RIFLE		AKIN		KDIGO		Urine Output
Criteria	Creatinine definition	Criteria	Creatinine definition	Criteria	Creatinine definition	
Risk	Serum creatinine increase: 1.5-fold; GFR decrease: >25%	Stage 1	Serum creatinine increase ≥ 0.3 mg/dL or ≥ 1.5 -fold increase from baseline within 48 hours	Stage 1	Serum creatinine increase ≥ 0.3 mg/dL within 48 hours or 1.5–1.9 times increase from baseline within 7 days	<0.5 mL/kg/h for >6 hours
Injury	Serum creatinine increase: 2-fold; GFR decrease: >50%	Stage 2	Serum creatinine ≥ 2 -fold increase from baseline hours	Stage 2	Serum creatinine 2.0–2.9 times increase from baseline within 7 days	<0.5 mL/kg/h for 12 hours
Failure	Serum creatinine increase: 3-fold; GFR decrease: >75%; Serum creatinine decrease: >350 μ mol/L (4 mg/dL) with acute increase >44 μ mol/L (0.5 mg/dL)	Stage 3	Serum creatinine increase ≥ 3 -fold from baseline or serum creatinine increase to ≥ 4.0 mg/dL with an acute increase of >0.5 mg/dL or institution of renal replacement therapy	Stage 3	Serum creatinine increased ≥ 3 -fold from baseline within 7 days or serum creatinine increase to ≥ 4.0 mg/dL with an acute increase of >0.5 mg/dL or institution of renal replacement therapy	<0.3 mL/kg/h for >12 hours
L, Loss	Persistent AKI = Complete loss of renal function for >4 weeks					
E, End stage	ESRD = Complete loss of renal function for >3 months					

(AKIN: Acute Kidney Injury Network; KDIGO: Kidney Disease Improving Global Outcomes; RIFLE: Risk, Injury, Failure, Loss and End-Stage Renal Disease)

AKIN broadens the risk category of RIFLE by taking into consideration an increase of ≥ 0.3 mg/dL of creatinine even though it may not be a 1.5-times increase in creatinine as per the RIFLE criteria. RIFLE required estimation of GFR as per the Modification of Diet in Renal Disease (MDRD) formula. This was removed from AKIN and creatinine values were used as there is no definitive way of measuring GFR and methods of estimation cannot be used in AKI. RIFLE criteria required creatinine to be assessed between 1 and 7 days, whereas with AKIN creatinine is measured within 48 hours; therefore, this would miss a slow-developing AKI over a 7-day period. AKIN criteria should be used only after the patient is volume resuscitated and reversible causes such as urinary tract obstruction are ruled out. KDIGO was built on both RIFLE and AKIN criteria in 2012. It includes a rise of ≥ 0.3 mg/dL creatinine within 48 hours like AKIN but assesses over a period of 7 days.

SUGGESTED READING

1. Ancona E, Rigotti P, Zaninotto G, Comandella MG, Morpurgo E, Costantini M. Treatment of lymphocele following renal transplantation by laparoscopic surgery. *Int Surg.* 1991;76:261-3.
2. Bucurescu G. (2018). Neurological manifestations of uremic encephalopathy. [online] Available from <http://emedicine.medscape.com/article/1135651-overview>. [Last accessed February, 2020]
3. Courcoulas AP, Kelly E, Harbrecht BG. Laparoscopic cholecystectomy in the transplant population. *Surg Endosc.* 1996;10:516-9.
4. Craig RG, Hunter JM. Recent developments in the perioperative management of adult patients with chronic kidney disease. *Br J Anaesth.* 2008;101:296-310.
5. De Boer J, De Meester J, Smith JM, Groenewoud AF, Bok A, van der Velde O, et al. Eurotransplant randomized multicenter kidney graft preservation study comparing HTK with UW and Euro-Collins. *Transpl Int.* 1999;12(6): 447-53.
6. Eilers H, Liu KD, Gruber A, Niemann CU. Chronic kidney disease: implications for the perioperative period. *Minerva Anesthesiol.* 2010;76:725-36.
7. Goyal VK, Solanki SL, Baj B. Pulmonary hypertension and postoperative outcome in renal transplant: a retrospective analysis of 170 patients. *Indian J Anaesth.* 2018;62:131-5.
8. Hutton P, Coopon G, James III F, Butterworth IVJ (Eds). *Fundamentals Principles and Practice of Anaesthesia*. Martin Dunitz: London; 2002.
9. Jankovic Z, Sri-Chandana C. Anaesthesia for renal transplant: recent developments and recommendations. *Curr Anaesth Crit Care.* 2008;19:247-53.
10. Jhaveri JK, Tan G, Scher DS, Tewari AK. Robot-assisted laparoscopic radical prostatectomy in the renal allograft transplant recipient. *J Endourol.* 2008;22:2475-9.
11. Juraj S, Kapural L, Bourke DL, O'Hara JF Jr. Anesthesia for kidney transplant surgery. *Anesth Clin Nor Am.* 2000;18: 919-51.
12. Kostopanagiotou G, Smyrniotis V, Arkadopoulos N, Theodoraki K, Papadimitriou L, Papadimitriou J. Anesthetic

- and perioperative management of adult transplant recipients in nontransplant surgery. *Anesth Analg*. 1999;89:613-22.
13. McDonald C, Milner Q. Chronic renal failure and anaesthesia. *Update anesth*. 2004;18(4). Available from https://www.wfsahq.org/components/com_virtual_library/media/f985c9bf9bf0c0e11a908bbf47fabae0-Chronic-Renal-Failure-and-Anaesthesia--Update-18-2004-.pdf. [Last accessed February, 2020]
 14. Nicholson G, Burrin JM, Hall GM. Perioperative steroid supplementation. *Anaesthesia*. 1998;53:1091-104.
 15. Paolucci V, Meyer W, Schaff B, Monico R. Laparoscopic drainage of a lymphocele after renal transplantation. *Surg Endosc*. 1994;8:111-3.
 16. Ploeg RJ, van Bockel JH, Langendijk PT, Groenewegen M, van der Woude FJ, Persijn GG, et al. Effect of preservation solution on results of cadaveric kidney transplantation. The European Multicentre Study Group. *Lancet*. 1992;340(8812):129-37.
 17. Robert G, Elkentaoui H, Pasticier G, Couzi L, Merville P, Ravaud A, et al. Laparoscopic radical prostatectomy in renal transplant recipients. *Urology*. 2009;74:683-7.
 18. Singh PM, Borle A, Makkar JK, Trisha A, Sinha A. Evaluation of transversus abdominis plane block for renal transplant recipients: a meta-analysis and trial sequential analysis of published studies. *Saudi J Anaesth*. 2018;12:261-71.
 19. Webb ST, Allen JSD. Perioperative renal protection. *Contin Educ Anaesth Crit Care Pain*. 2008;8(5):176-80.
 20. Zacharias M, Gilmore IC, Herbison GP, Sivalingam P, Walker RJ, Hovhannisyan K. Interventions for protecting renal function in the perioperative period. *Cochrane Database Syst Rev*. 2005;3:CD003590.

Tonsillectomy

Sheila Nainan Myatra, Sumitra Ganesh Bakshi, Jeson Rajan Doctor

A 4-year-old male child, weighing 16 kg has a history of 3–4 episodes of tonsillitis in the past year. He now presents with:

- *Throat pain and dysphagia which has progressively increased:* Since 10 days
- *Reduced intake:* Since 8 days
- *Fever:* Since 2 days
- *Severe weakness:* Since 2 days.

On examination the child is febrile, heart rate (HR)—120 beats/min, looks dehydrated and has enlarged and inflamed palatine tonsils. The child is posted for tonsillectomy due to repeated infections.

Q. What are tonsils? What are adenoids? What is their function? What is tonsillitis?

Ans. The tonsils are areas of lymphoid tissue on either side of the throat. Most commonly, the term “tonsils” refers to the palatine tonsils that can be seen in the back of the throat. There are three masses of tissue—(1) the tubal, (2) palatine, and (3) lingual tonsil. The adenoid tissue is positioned in the midline of the posterior nasopharyngeal wall immediately inferior to the rostrum of the sphenoid. Together this set of lymphatic tissue along with the adenoid is known as Waldeyer’s tonsillar ring.

The tonsils are located in the lateral oropharynx. The tonsillar branch of the facial artery forms the main arterial blood supply. The venous drainage is via a plexus surrounding the tonsil, which drains into the pharyngeal plexus. The external palatine vein enters the tonsillar bed from the soft palate. This large vein is usually responsible for the venous hemorrhage following tonsillectomy.

The sensory supply is from the glossopharyngeal and lesser palatine nerves. Important structures deep to the inferior pole of the tonsil are the glossopharyngeal nerve, the lingual artery, and the internal carotid artery.

Like other organs of the lymphatic system, the tonsils act as part of the immune system to help protect against infection. In particular, they are believed to be involved in helping fight off pharyngeal and upper respiratory tract infections. Tonsillitis is the infection of the tonsils.

Q. What are the indications and contraindications for tonsillectomy and adenoidectomy?

Ans. The indications for surgery may be absolute or relative.

Indications:

- Upper airway obstruction, dysphagia, and obstructive sleep apnea
- Peritonsillar abscess, not responding to adequate medical management and surgical drainage
- The requirement of biopsy to confirm tissue pathology in suspected neoplastic causes
- *Recurrent tonsillitis that is unresponsive to medical treatment:*
 - Sore throat secondary to tonsillitis
 - More than 5 episodes of tonsillitis in 1 year
 - Symptoms persisting for more than 1 year
 - Severe disabling episodes of sore throat.
- Persistent bad-breath and taste in mouth due to chronic tonsillitis
- Persistent tonsillitis in streptococcus carrier, which is unresponsive to antibiotics.

Contraindications:

- Presence of acute infection
- Abnormal coagulation profile
- Uncorrected partial/complete cleft palates in these patients adenoids help to fill the velopharyngeal gap and facilitate speech.

Q. What is the usual clinical presentation of children with tonsillitis?

Ans.

- Pain in the throat
- Dysphagia

- Mouth breathing
- Failure to thrive/repeated infection—pain fever, tachycardia
- Cervical adenopathy
- Visibility inflamed tonsil which may have discharge.

Q. What are the different methods available for tonsillectomy?

Ans.

- Blunt dissection and tonsillar snare
- Bipolar radiofrequency
- Electrocautery
- Ultrasonic dissection
- Laser
- Powered microdebrider.

Q. What history and clinical examination will you perform in this child?

Ans.

History

- Routine anesthetic history and examination for a pediatric patient including milestone development and vaccination
- *History of tonsillitis*: Repeat episodes of fever, throat pain, and dysphagia
- History of any easy bruising, bleeding gums, epistaxis, menorrhagia or bleeding at any other sites
- Family history of any bleeding disorders
- Recent ingestion of aspirin, nonsteroidal anti-inflammatory drugs (NSAIDs)
- Mouth breathing
- The triad of hyponasality, snoring, and mouth breathing normally indicates enlarged, obstructing adenoids
- Other symptoms of adenoid disease include rhinorrhea, postnasal drip, chronic cough, and headache
- History of possible allergies, gastroesophageal reflux disease (GERD), and sinusitis
- *Obstructive sleep apnea*: In children, adenotonsillar hypertrophy is the most common cause of obstructive sleep apnea. The signs and symptoms include chronic hypoxemia manifesting itself as polycythemia and right ventricular strain. The symptoms like snoring, apneic episodes followed by grunting and restlessness occurring during sleep. The daytime symptoms include headaches, excessive daytime somnolence, and not feeling fresh in the morning.

Examination

- Routine examination in a pediatric patient
- *Loose/missing teeth*: Most children have fallen milk teeth at this time. This is also important for application of mouth gag
- Patency of oral and nasal cavity

- Patients may have “adenoid facies” (long face, flattened midface, open mouth) and hyponasal speech
- Enlarged (>2 cm) or tender cervical adenopathy
- Tonsillar or pharyngeal exudates.

Q. What investigations will you ask for?

Ans.

- Hemoglobin and complete blood count (CBC)
- Coagulation profile and platelets only if there is history suggestive of bleeding tendencies.

Q. How will you optimize this child prior to surgery?

Ans. This child has clinical signs suggestive of infection and dehydration probably because of inadequate intake due to dysphagia. Hence, a course of antibiotics should be started and adequate hydration in the form of intravenous (IV) fluids should be given prior to surgery.

Q. How will you premedicate this child?

Ans. Premedication should include an antisialogogue and possibly a narcotic. However, barbiturates will be of little use in short upper airway surgery which requires quick return of protective airway reflexes. Sedatives should not be used if there is history suggestive of obstructive sleep apnea.

Q. What are the anesthetic considerations for tonsillectomy surgery?

Ans. Basic principles of anesthesia for tonsillectomy in children remain the same as for general pediatric anesthesia. Specific points with reference to intraoperative and postoperative management are described below:

- The goal of anesthesia for elective tonsillectomy is to maintain deep general anesthesia (GA) that prevents reflex-induced hypertension, tachycardia or arrhythmias. Muscle relaxation is required to allow placement of the mouth gag and prevent any bucking, coughing or straining. A rapid recovery of consciousness and return of protective airway reflexes is also desired.
- Inhalational induction with sevoflurane is preferred in small children, especially when IV line is not inserted, and in obstructive sleep apnea (OSA) patients. Parental presence during anesthetic induction may be a useful technique to make the experience more pleasurable and avoiding parental separation anxiety. If an IV line is present propofol or ketamine may be given in sedation doses in the presence of the parent and the child wheeled into the operating room with a patent airway and supplemental oxygen under close monitoring. Intramuscular injections should be discouraged. Various nonpharmacological techniques have also been described such as toys, videos, clown doctors, distraction techniques, toy cars, etc. for avoiding or minimizing separation anxiety from the parents.

- **Endotracheal intubation:** Intubation under deep inhalational or muscle relaxant assisted anesthesia is preferred. Regular tube/RAE tube may be passed by orotracheal route. Throat should be well packed especially when uncuffed tubes are used to prevent aspiration of blood and secretions. Microcuff tubes have a distally placed polyurethane cuff and are high-volume low-pressure cuffed tubes and are becoming increasingly popular for pediatric use. An oral endotracheal tube (ETT) can either be fixed in the midline or fixed on one side at the angle of the mouth and the side changed once the tonsillectomy is done and hemostasis achieved for removal of the opposite tonsil. When only tonsillectomy and no adenoidectomy is planned one can also insert a nasotracheal tube. Intubation could be difficult if the tonsils are very large and approximating in the midline (kissing tonsils).
- Flexible laryngeal mask airway (LMA) may be used for adenotonsillectomy surgeries and is practiced in some centers by experts. However, we do not recommend its use for this surgery. The advantages of LMAs are that they require a lighter plane of anesthesia, and there is no need for muscle relaxants to be used with resultant rapid induction and smooth recovery. The LMA is not removed until full return of reflexes. The main disadvantage is if it gets dislodged, the airway may be lost during surgery, leading to avoidable complications and a situation that may become difficult to rectify. Thus endotracheal intubation remains the gold standard for tonsillectomy surgery.
- **Maintenance:** Anesthesia is maintained with inhaled anesthetics and short-acting opioids like fentanyl using spontaneous ventilation and/or muscle relaxants with controlled ventilation. However, adequate depth should be maintained to prevent any reflex-induced hypertension, tachycardia and arrhythmias, and avoid bucking, coughing or straining during surgery.
- Local anesthetic plus adrenaline applied in the tonsillar fossa gives the advantages of bloodless dissection, reduced operative time, and reduced postoperative pain. If large volumes are injected, it can give rise to respiratory obstruction once the patient is extubated because of bilateral glossopharyngeal nerve block.
- Since the operative field is close to the airway, one should be very vigilant about accidental extubation or aspiration of blood and secretion if the throat pack is displaced under GA when uncuffed tubes are used. Microcuffed tubes will be helpful in preventing trickling of oral secretions and blood into the airway.
- Blood loss during tonsillectomy may be difficult to estimate and may reach up to 5% of the blood volume.

Blood transfusion may be required in some cases. IV fluids like Ringer's lactate (RL) are balanced crystalloids and are recommended to be used to maintain adequate volume status.

- Protection of the eyes is very important especially when the gag is used.
- At the end of surgery, pack removal and good pharyngeal and laryngeal suction under vision is essential.
- An antiemetic should be given prior to reversal. Patients undergoing tonsillectomy are prone to develop postoperative nausea and vomiting (PONV), so an antiemetic is recommended: ondansetron (0.1 mg/kg) or dexamethasone (0.1–0.2 mg/kg) or a combination of both can be considered.
- **Reversal:** Reversal of muscle relaxant should be done using neostigmine and atropine and patient should be extubated only when awake and there is return of protective airway reflexes. Extubation should be smooth thereby preventing rise in blood pressure which can cause bleeding.
- Adequate analgesia is essential to have a smooth extubation.
- Patient should be transported in tonsillar position with oxygen supplementation.

Q. How is the patient positioned during surgery?

Ans. The patient's head is put in as lightly extended position, held by as and bag/bolster, under the shoulder, while the mouth is kept open and the tongue is held out of the way by a Boyle–Davis or a similar gag. In this position, the operator has a direct view of the tonsils and there is the added advantage of the posterior part of the pharynx forming a sump into which the blood may drain, below the level of the glottis.

It is important to check that the ETT has not migrated out during extension of the neck.

Q. How will you manage postoperative pain after tonsillectomy?

Ans. Post-tonsillectomy pain is usually mild-to-moderate and can be taken care of by paracetamol syrup. NSAIDs are usually avoided because of their tendency to increase bleeding and cause bronchospasm in patients with allergic diathesis. Intraoperative infiltration of the tonsillar bed with local anesthetic, spraying the tonsillar fossa with lignocaine spray, paracetamol IV, IV opioids, IV ketorolac, ibuprofen, and paracetamol syrup, intranasal sprays or transmucosal fentanyl are some of the analgesic techniques described in literature for postoperative analgesia for tonsillectomy. Adequate analgesia makes the child calm and maintains the hemodynamics and decreases the incidence of post-tonsillectomy bleed. However, it should be remembered

that the child should be fully awake to prevent aspiration of secretions and blood in the event of a rebleed. The risks and benefits of using NSAIDs for analgesia should be individualized based on the patient.

Q. What is the tonsillar position? What is its clinical significance?

Ans. After surgery the child is placed in the "tonsil position" which is a left lateral position, with one knee flexed and the hand under the face along with a slight head low position. This allows the blood and secretion to drain out rather than flow back onto the vocal cords.

Q. What are the complications you can anticipate in the immediate postoperative period?

Ans.

- Post-tonsillectomy bleeding
- Airway obstruction because of upper airway edema, presence of blood and secretions and laryngospasm
- Postoperative nausea and vomiting during first 24 hours (as high as 70%) because of pharyngeal mucosal irritation from surgery and swallowed blood and secretions
- Pain and sore throat lasts for 3–4 days
- Postoperative respiratory complications
- Negative pressure pulmonary edema may occur due to sudden release of upper airway obstruction, but this is very rare.

Q. How do you diagnose post-tonsillectomy bleed? How do you plan the anesthetic management?

Ans. Bleeding is seen more in older children (age >11 years >69%) and in patients with chronic tonsillitis (75%).

Primary bleeding occurs within first 24 hours, secondary bleeding from 24 hours to 5–28 days. Bleeding from adenoidal bed (3.2%) is more common in first 4 hours, tonsillar bleeding (1.2%) is more common in first 6–8 hours. For day care procedures, it is safer to observe the patient for 8–10 hours after surgery. The incidence of bleeding following tonsillectomy is 0.5–2% depending upon the surgical technique.

Secondary bleeding: This is associated with sloughing of the Eschar (dead tissue) overlying the tonsillar bed, loosened vessel ties or infection from underlying chronic tonsillitis.

Any amount of bright red blood coming from the mouth or the nose should alert the doctor to the possibility of postoperative bleeding. Frequent swallowing, tachycardia, and coffee-ground emesis are indirect signs of bleeding. If the patient complains of intermittent bleeding but is not actively bleeding on presentation, he or she should be admitted for overnight observation and administration of IV fluids. Coagulation testing should be obtained following a bleeding episode, and if abnormal, hematology consult and

additional coagulation testing should be obtained. A CBC should also be obtained to assess the degree of hemorrhage and as a baseline in case of further bleeding.

Minor bleeding may be controlled by gargling with ice water and packing. If bleeding persists and the patient is uncooperative, he will be needed to be taken up for re-exploration under anesthesia. Important considerations while anesthetizing a child for control of bleeding after tonsillectomy are:

- Degree of blood loss may be underestimated, as it is not visible and it is swallowed.
- Child may lose large amounts of blood and become hypovolemic and even progress to shock in a short time. Child may even have altered consciousness if bleeding is severe.
- Immediate resuscitation with colloid and crystalloid while waiting for blood to become available. More blood should be kept cross-matched for surgery.
- Preoperative sedation should be avoided.
- Adequate preoxygenation.
- Wide bore good IV access.
- Intravenous induction agents depending on hemodynamic stability (thiopentone sodium, propofol) may be used.
- Child should be considered as full stomach as large amount of blood and secretions may be swallowed. A rapid sequence intubation with cricoid pressure and cuffed ETT using succinylcholine is warranted. Two good working suctions should be ready at the head end in case of vomiting.
- Reintubation may be difficult if bleeding is obscuring the view or due to edema from previous airway instrumentation and surgery. A smaller size ETT than the previous anesthetic should be ready. This may be needed due to presence of airway edema.
- The child may become hypothermic during surgery due to large volume transfusion. The child should be kept well covered to maintain body temperature, and if possible a warming blanket used with temperature monitoring. Hypothermia exacerbates coagulopathy.
- Decompression of stomach should be done with a nasogastric tube after intubation, to ensure that the stomach is empty prior to extubation.
- Extubation should be done in lateral position and only if the child is fully awake with normal gag, cough reflex and is stable hemodynamically.

Q. How is the anesthetic management of tonsillectomy in an adult different from children?

Ans. This is managed exactly in the same way as in children, except that, as adenoid tissue is not present in adults the tracheal tube is more often passed nasally than through the

mouth. Also the requirements of postoperative analgesics and antiemetics are greater in adults than in children.

Q. Is there a role for performing tonsillectomy under local anesthesia?

Ans. Due to the multiple origins of the nerve supply to the tonsil, there is no single nerve trunk that can be blocked to give effective anesthesia. However, local infiltration around the capsule is quite effective. Following topical anesthesia of the mucous membrane, three separate injections of local anesthetic solutions are made—one in the region of the upper pole, one into the anterior pillar, and one in the region of the lower pole. In each instance, attempt is made to deposit the solution between the capsule and the pharyngeal wall:

- This is a very vascular and special care must be taken not to inject local anesthetic into a blood vessel.
- Tonsillectomy under these circumstances is, however, unpleasant for the patients and surgeons alike.
- Small children will not be cooperative for local injections.
- It may also be dangerous in situations where the protective laryngeal reflexes can be obtunded by spread of the nerve block beyond the immediate area of the tonsil making even a conscious patient liable to laryngeal obstruction and aspiration of blood and debris.

For these reasons tonsillectomy is rarely performed under local anesthesia. However, the use of local infiltration anesthesia in combination with GA has been claimed to reduce the reflex response from surgical field, reduce bleeding, aid dissection and contribute to postoperative comfort.

Q. How will you manage a case of peritonsillar abscess or quinsy?

Ans. This is more frequently seen in older children. This occurs in proximity to the tonsil, but outside the tonsillar capsule and most often in the soft palate. It is usually limited to one side and causes severe sore throat, difficulty in swallowing, high fever and progressive difficulty in

opening the mouth. The affected tonsillar area is markedly inflamed with uvula displaced to the opposite side. If it does not resolve with antibiotics, then incision and drainage into the pharynx will be necessary. Older child may allow the drainage under conscious sedation but for anxious or smaller children, GA would be needed.

Consideration while Administering General Anesthesia

- Aggravation of a preexisting respiratory obstruction.
- Even with relaxation, trismus may not resolve, making laryngoscopy and intubation difficult.
- Abscess may rupture at any time during induction or intubation and there is a risk of aspiration of purulent material.
- General anesthesia is induced with inhalational agent in oxygen or IV induction agents such as propofol along with sevoflurane.
- Patient is kept in head low position with the head turned toward the affected side.
- Under deep plane of anesthesia laryngoscopy is done extremely carefully for fear of rupturing the abscess keeping two wide bore suction ready.
- Muscle relaxant should not be given until the anesthesiologist is certain about securing and maintain the airway. Once airway is secured with cuffed ETT, the rest of the management is straight forward.

SUGGESTED READING

1. Davies K. Anaesthesia for tonsillectomy. *Anaesthesia Tutorial of The Week* 51. 2007. pp. 1-9.
2. Davis PJ, Cladis FP, Motoyama EK. *Smith's Anaesthesia for Infants and Children*, 8th edition. Philadelphia, USA: Elsevier Mosby; 2011.
3. Hermans V, De Pooter F, De Groote F, De Hert S, Van der Linden P. Effect of dexamethasone on nausea, vomiting, and pain in paediatric tonsillectomy. *Br J Anaesth*. 2012;109:427-31.
4. Ravi R, Howell T. Anaesthesia for paediatric ear, nose, and throat surgery. *Contin Educ Anaesth Crit Care Pain*. 2007;7:33-7.
5. Sethi D, Smith J. Anaesthesia for Bleeding Tonsil. [online] Available from <https://www.frca.co.uk/Documents/109%20Anaesthesia%20for%20the%20bleeding%20tonsil.pdf> [Last accessed February, 2020].

Circumcision

Parmanand Jain, Sudivya Sharma

A 1-year-old child, has ballooning and pain around penis during micturition, is scheduled for circumcision.

Q. What are the preoperative considerations in circumcision?

Ans. It is a common day-case minor procedure of about 20 minutes, with minimal blood loss, and it entails removal of prepuce or preputioplasty or prepuce plasty.

Q. What are the various anesthesia techniques used for circumcision?

Ans. It can be done under general anesthesia (GA) with spontaneous ventilation [with laryngeal mask airway (LMA)], caudal or penile block or ring block.

Q. How do you control postoperative pain?

Ans. It is a very painful day-case procedure and parents are advised to apply topical lignocaine jelly regularly and diclofenac suppository (1 mg/kg) >1 year or paracetamol suppository 30–40 mg/kg <1 year of age for several days.

Q. How do you give penile block?

Ans. The pudendal nerve (S2–S4) divides into the right and left dorsal nerves of the penis those pass under the pubis symphysis and travel below Buck's fascia (deep fascia of the penis) to supply sensory innervation to the penis. Previous studies have reported failure rates of around 4–6.7%, using the dorsal nerve block. So far, penile nerve block is the most popular nerve block for the circumcision in pediatric patients (**Fig. 1**).

The safest technique is to inject an adequate volume of local anesthetic bilaterally deep to the fascia into the pear-shaped spaces on each side of the suspensory ligament. This avoids midline injection and therefore potential damage to the dorsal vessels and provides the maximum chance of diffusion into the nerves to block them. Injecting an adequate volume of local anesthetic (estimated in children

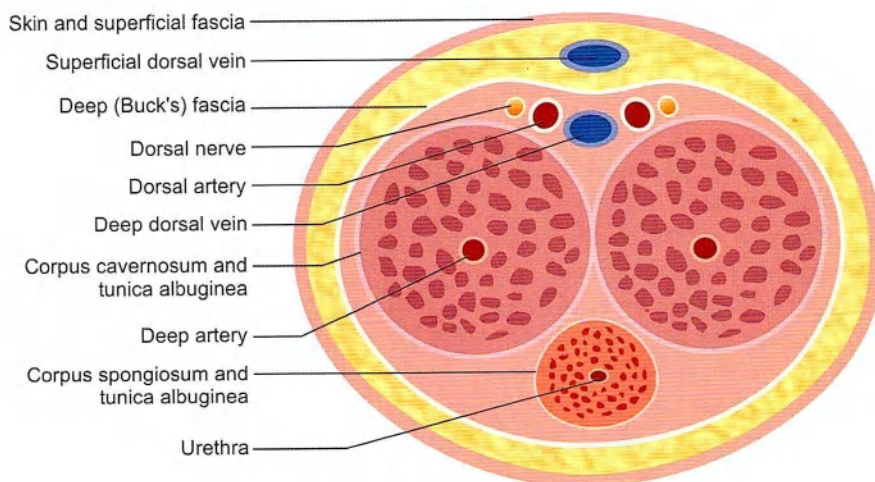


Fig. 1: Dorsal penile nerve block.

at 1 mL + 0.1 mL/kg on each side) should ensure that the ventral branch is also blocked so that a satisfactory block is achieved.

The technique involves inserting the needle until it touches the pubic symphysis. This gives a guide to depth. The needle is then withdrawn and redirected to pass below the symphysis and 3–5 mm deeper depending on the size of the patient. It is preferable to direct it slightly laterally into the pear-shaped space and then to reinsert in on the other side depositing equal volumes on each side. Avoiding the midline injection reduces the chance of penetrating the dorsal vessels of the penis and causing hematoma. If a short beveled needle the fascia may be felt as a slight resistance when it is penetrated, but in small children, this is not always felt as it is thin and may offer little resistance.

Q. Describe the caudal block.

Ans.

Anatomy: The sacrum articulates with the fifth lumbar vertebra above and the coccyx below. The sacral hiatus is shaped by incomplete midline fusion of the posterior elements of the distal portion of the fifth or sometimes the fourth sacral vertebra. This inverted U-shaped space is covered by the posterior aspect of the sacrococcygeal membrane and is an important landmark in caudal epidural block. The remnants of the inferior articular process elongate downward on both sides of the sacral hiatus. These two bony processes are called the sacral cornua (horns) and define important clinical landmarks during caudal epidural block. The hiatus is covered only by skin, a subcutaneous fatty layer and the sacrococcygeal membrane. The distal-most portion of the dural sac and the sacral hiatus usually terminates between levels S1 and S3.

The sacral canal contains:

- The terminal part of the dural sac, ending between S1 and S3.
- The five sacral nerves and coccygeal nerves making up the cauda equina. The sacral epidural veins generally end at S4, but may extend throughout the canal. They are at risk from catheter or needle puncture.
- *The filum terminale:* The final part of the spinal cord which does not contain nerves. This exits through the sacral hiatus and is attached to the back of the coccyx.
- Epidural fat, the character of which changes from a loose texture in children to a more fibrous close-meshed texture in adults. It is this difference that gives rise to the predictability of caudal local anesthetic spread in children and its unpredictability in adults.

Technique of caudal block: Aseptic technique is mandatory. Position of the patient in the left lateral position with the legs flexed at the hip. The flexed arch of the child not only helps to palpate the sacral cornua better, but also shifts the

end of dural sac cranially. Identify the sacral hiatus as the apex of an equilateral triangle with the base formed by a line joining the posterior superior iliac spines. The sacral hiatus is covered by the sacrococcygeal membrane. Make a small nick in the skin with a needle to reduce the possibility of a dermoid. Use a 22-gauge or 20-gauge cannula at 60° to the skin from the midpoint of the line joining the sacral cornua. Short beveled needles with a stylet which are specially designed for caudal blocks are also available, e.g., SPROTTE cannula, Crawford cannula, etc. A small “give” indicates penetration of this membrane. Flatten the cannula and then advance, not >1–3 mm. Advancement of a cannula rather than needle may reduce the incidence of inadvertent dural or vascular puncture. The distance between the end of dural sac and puncture site may be only a few millimeters in small infants. Test aspiration should be gentle. Aspiration should be repeated during injection of local anesthetic. 0.25% bupivacaine is commonly administered (0.5–1 mL/kg). The analgesic effect of long-acting local anesthetic agents like bupivacaine lasts for 4–8 hours.

A 2008 Cochrane review showed no difference in pain scores between caudal block and dorsal penile block, but described more motor block with caudal block.

Complications of caudal block: Subcutaneous injection (visible swelling), superficial skin infection, intrathecal injection, intravascular injection, local anesthetic toxicity, back pain, urinary retention, sacral osteomyelitis, etc.

Recent advances:

- Ropivacaine 0.2% is a relatively safer local anesthetic with better cardiovascular profile and intrinsic vasoconstrictor property. It has beneficial ratio of sensory to motor block.
- Levobupivacaine, a new long-acting amide-type local anesthetic, is the S(–)-isomer of the racemate bupivacaine. It is safer than bupivacaine and efficacy is similar.
- The technique can be made more accurate and safe with the use of ultrasound to guide and confirm needle placement and also see the spread of the drug. Posterior dural sag can also be seen as the injected drug moves the posterior dura anteriorly. Ultrasound usage has an additional advantage in infants as neural conduit can be better visualized as ossification of spine is incomplete.
- Continuous catheter technique can be used by inserting catheter through the cannula. The tip of the catheter can be confirmed using X-ray or ultrasound.
- Few anesthetists prefer tunneling of catheters as being near the perineum, it is prone to get soiled and get infected.
- Electrostimulation with insulated needles to see end motor response, i.e., contractions of anal sphincter (S2–S4).

This can even distinguish intrathecal placement as much smaller current (<1 mA) is required.

Adjuvants used: These are agents used with local anesthetic drugs to improve quality of block in terms of duration of block, onset, recovery and density of block. The examples are epinephrine (5 µg/mL), opioids like morphine (0.05 mg/kg), fentanyl (1 µg/kg), buprenorphine (0.03 mg/kg), preservative-free ketamine (0.5 mg/kg), clonidine (1 µg/kg), midazolam (50 µg/kg), etc.

Q. What are the recent updates in regional anesthesia used for circumcision?

Ans. Recently few studies have compared penile nerve block with pudendal nerve block, suggesting significantly lower postoperative pain intensity and analgesic consumption in the pudendal block.

Pudendal nerve block can be performed using surface landmarks, with nerve stimulation or with ultrasound guidance. The pudendal nerve arises from the sacral plexus with contributions from the ventral rami of S2–S4. It exits through the greater sciatic foramina inferior to the piriformis muscle, crosses the posterior aspect of the sacrospinous ligament at the level of the ischial spine, and reenters the pelvis through the lesser sciatic foramina. It then travels posteroinferiorly through pudendal canal and divides into the inferior rectal nerve, perineal nerve, and dorsal nerve of the clitoris or penis.

The ischial spine is the landmark. Local anesthetic is typically injected posterior to the ischial spine at the attachment of the sacrospinous ligament. Injecting anesthetic medial to the ischial spine maximizes the distance between the needle trajectory and important structures such as nerves and the pudendal artery.

SUGGESTED READING

1. Adewale L, Dearlove O, Wilson B, Hindle K, Robinson DN. The caudal canal in children: a study using magnetic resonance imaging. *Pediatr Anesth*. 2000;10:137-41.
2. de Beer DA, Thomas ML. Caudal additives in children—solutions or problems? *Br J Anaesth*. 2003;90(4):487-98.
3. Holve RL, Bromberger PJ, Groveman HD, Klauber MR, Dixon SD, Snyder JM. Regional anesthesia during newborn circumcision. *Clin Pediatr (Phila)*. 1983;22:813-8.
4. Kalava A, Pribish AM, Wiegand LR. Pudendal nerve blocks in men undergoing urethroplasty: a case series. *Rom J Anaesth Intensive Care*. 2017;24:159-62.
5. Kirya C, Werthmann MW. Neonatal circumcision and dorsal penile nerve block—a painless procedure. *J Pediatr*. 1978;92:998-1000.
6. Maxwell LG, Yaster M, Wetzel RC, Niebyl JR. Penile nerve block for newborn circumcision. *Obstet Gynecol*. 1987;70:415-9.
7. Naja Z, Al-Tannir MA, Faysal W, Daoud N, Ziade F, El-Rajab M. A comparison of pudendal block vs dorsal penile nerve block for circumcision in children: a randomised controlled trial. *Anaesthesia*. 2011;66(9):802-7.
8. Poma PA. Painless neonatal circumcision. *Int J Gynaecol Obstet*. 1980;18:308-9.
9. Ponde VC. Recent developments in paediatric neuraxial blocks. *Indian J Anaesth*. 2012;56(5):470-8.
10. Porter FL, Miller RH, Marshall RE. Neonatal pain cries: Effect of circumcision on acoustic features and perceived urgency. *Child Dev*. 1986;57:790-802.
11. Sara CA, Lowry CJ. A complication of circumcision and dorsal nerve block of the penis. *Anaesth Intensive Care*. 1985;13:79-82.
12. Talbert LM, Kraybill EN, Potter HD. Adrenal cortical response to circumcision in the neonate. *Obstet Gynecol*. 1976;48:208-10.
13. Tutuncu AC, Kendigelen P, Ashyeralyeva G, Altintas F, Emre S, Ozcan R, et al. Pudendal nerve block versus penile nerve block in children undergoing circumcision. *Urol J*. 2018;15(3):109-15.

Acute Postoperative Pain

Parmanand Jain, Sumitra Ganesh Bakshi, Malini Joshi

The International Association for the Study of Pain has proposed a working definition for pain: Pain is an unpleasant sensory and emotional experience associated with either actual or potential tissue damage.

During the past two decades, undertreatment of pain has been recognized as an important issue in healthcare worldwide. Deficiencies in pain management maybe due to inadequate staff training, knowledge deficits, unhelpful staff, patient attitudes, poor pain assessment, fear of analgesic side effects, and lack of accountability. Many hospitals have multidisciplinary acute pain services (APS) for management of postoperative pain, training, development of guidelines, and to undertake research and audit which leads to improved care in pain management. Also, the requirement of a dedicated acute pain nurse for effective pain service has been emphasized. Specialized pain relief methods such as patient-controlled analgesia (PCA) and epidural and perineural infusions, now included in APS, have improved care. However, many patients still continue to suffer considerable pain. It is essential to lay down protocols for postoperative pain management in accordance with the nature of the surgery and severity of pain anticipated in the postoperative period.

Q. What are the causes of postoperative pain? Why is it important to treat it?

Ans. Surgical procedures invariably cause tissue damage along with presence of drains, tubes, and postoperative complications resulting in pain. Postoperative pain is often underestimated and undertreated. The impact of inadequate pain relief can result in delayed mobilization, increased morbidity and mortality due to respiratory and thromboembolic complications, increased hospital stay, psychological distress, and anxiety. Pain management in a perioperative setting refers to actions before, during, and after a procedure that is intended to reduce or eliminate postoperative pain before discharge.

Q. Describe the pathophysiology of postoperative pain. Explain the concept of chronic postsurgical pain and how will you manage it. Why is it difficult to treat postoperative pain?

Ans. Studies have shown that incisional pain differs from inflammatory or neuropathic pain. Hyperalgesia in incisional pain maybe mediated by sensitization of A fiber and C fiber nociceptors and conversion of silent A- δ nociceptors to mechanically sensitive fibers after incision. Ischemic pain mechanism may also contribute to postsurgical postoperative pain as indicated by increased

lactate concentration and low pH occurring in skin and muscle wounds. Central neuronal sensitization may also be a contributory factor.

Chronic postsurgical pain is more common, especially after certain types of surgery, for example, thoracotomy and mastectomy. It should be diagnosed when pain persists beyond the expected healing period associated with tissue injury (2 months or longer after most surgical procedures). Ongoing inflammation or injury to peripheral nerves may lead to persistent pain. Predictive factors for developing continuing postoperative pain include preoperative pain, repeat surgery, prolonged surgery, severe postoperative pain, surgical approaches with a higher risk of nerve damage, chemotherapy or radiation, and psychological and depressive symptoms. Pre-emptive analgesia in which one or more analgesic is administered prior to a noxious event to prevent peripheral and central sensitization reduces postinjury pain. There is some evidence that it prevents development of chronic pain, but it is highly likely that early intervention when signs are detected is more likely to be beneficial. A pre-emptive analgesic effect maybe influenced by multiple factors such as choice of drug, nature and degree of tissue damage, duration of surgery, time, route of administration, and time period of central sensitization.

Q. Enumerate the common drugs used for postoperative pain management.

Ans. Acute pain can cause pathophysiological responses, which are initiated when nociceptors are activated after tissue injury causing local inflammatory response, and behavioral and neuroendocrine activation like tachycardia, hypertension, hyperglycemia, immunosuppression, decreased regional blood flow, venous stasis, and platelet aggregation.

Pharmacologic treatment is the mainstay. Multimodal analgesia in the form of paracetamol, nonsteroidal anti-inflammatory drugs (NSAIDs), and opioids and modern techniques such as "patient-controlled analgesia" (PCA), epidural, and perineural infusions are effective in treating postoperative pain.

Q. What is the rationale for multimodal analgesia?

Ans. Multimodal analgesia uses a combination of two or more analgesics with an aim to improve analgesia through the use of analgesics, each with a different mode of action, and to decrease side effects through a reduction in the doses of analgesics, particularly opioids. It can be defined as a combination of opioid and nonopioid analgesics with or without a regional anesthetic block, resulting in improved analgesia with decrease in side effects. Studies have compared patients with intravenous (IV) PCA with and without additional agents and have measured outcomes such as efficacy, opioid consumption, side effects, and complications. One study examined the addition of paracetamol, selective cyclo-oxygenase-2 inhibitors (COX-2 inhibitors), and NSAIDs to IV PCA revealing that all provided an opioid-sparing effect. The morphine-sparing effect with paracetamol was <20%, with COX-2 inhibitors ~25% and with multiple doses of NSAIDs ~40%. Whenever possible, an anesthesiologist should use multimodal pain management therapy. Central regional blockade with local anesthetics should be considered. Unless contraindicated, patients should receive a round-the-clock regimen of COXIBs, NSAIDs, or acetaminophen. Dosing regimens should be administered to optimize efficacy while minimizing the risk of adverse events. The choice of medication, dose, route, and duration of therapy should be individualized.

Q. What is the role of paracetamol (acetaminophen)?

Ans. Paracetamol has been widely accepted as an effective analgesic and it is used in combination with other analgesics for moderate or severe pain. Further, IV paracetamol due to its quick onset and efficacy ensures a more predictable plasma concentration and may provide considerable opioid-sparing effect. Many prescriptions now include paracetamol to be given regularly by the clock and often via the IV route for the immediate postoperative period due to its lower side effects, better profile, and tolerability.

For healthy adults, the daily dosing should be limited to 4 g/day. As the bioavailability from the oral route is good, one must switch from IV to oral route.

Q. What is the role of NSAIDs?

Ans. The NSAIDs are the most commonly used postoperative analgesic drugs including COX-2 inhibitors (aspirin, diclofenac, and ibuprofen) and new COX-2 selective inhibitors. They are effective in postoperative pain; however, adverse effects and contraindications may limit their use. The main concerns are interference with platelet function, renal impairment, gastric and duodenal ulceration, and potential for bronchospasm in asthmatics sensitive to aspirin and NSAIDs. Careful patient selection is necessary. NSAIDs being peripherally acting analgesics improve analgesia significantly, attenuate hyperalgesia around the incision, and reduce the incidence of nausea, vomiting, and sedation when used in conjunction with PCA. There is a considerable opioid-sparing effect and may benefit patients in whom low opioid doses are preferred.

Q. What is PCA? What are the other modes of analgesia?

Ans. Patient-controlled analgesia is essentially a system wherein the patient is enabled to self-administer a preset amount of analgesic drug as and when required. This system is commonly used for the IV route, though it can also be used for epidural [patient controlled epidural analgesia (PCEA)], intranasal, subcutaneous, transdermal, and regional infusions. It is essentially an infusion pump/portable machine with a push button which delivers the drug when triggered. PCA improves patient satisfaction, as it allows the patient to independently titrate his/her analgesic requirements allowing him/her greater control over his/her treatment.

When used through the IV route, opioids are the drugs administered through this system. Absence of an upper daily limit and metabolites devoid of organ toxicity makes opioid the preferred drug. Morphine or fentanyl is commonly used in our subcontinent.

Although PCA is mostly indicated for postoperative analgesia, it can be also used to maintain pain relief once pain control is achieved, e.g., acute pancreatitis, rib fractures, or trauma awaiting surgery. It is important to remember that PCA is essentially a maintenance therapy; therefore before starting the PCA (individually titrated doses), the patient's pain should be initially controlled by giving analgesic boluses.

Patient-controlled analgesia is not indicated in elderly, confused patients, preschool children, patients with poorly functioning hands (arthritis, hand surgery, spinal injury) who may not be able to press the demand button, or the very young who may not be suitable candidates. Nurse-based or caretaker-based PCA can be used in these

patients. Although PCA is a relatively simple concept, its use requires dynamic interaction with both the patient and the machine, so a preoperative assessment as to the willingness of the patient to use PCA should be conducted. The patient must be counseled about its correct use and he/she must be comfortable to use it independently without fear of overdose and addiction. Preoperatively, pain-related counseling must be done by the anesthetist responsible for the patient. Subsequently, the ward and recovery nurses should reinforce the education. Errors may include programming errors and faulty equipment. Staff training and safe standardized systems, including monitoring requirements, are essential.

Postoperative nausea and vomiting, which is a side effect of opioids, must be monitored in these cases. It is important to have effective communication, education, and careful attention to reduce side effects. Considering a change to an alternative opioid, if the patient has persistent side effects, is also important.

Q. What is the role of regional anesthesia in postoperative pain?

Ans. Regional anesthesia is now included as a valuable component of the multimodal combination. It can be used as local wound infiltration or infusions, perineural injections, spinals, and epidurals. Wound infiltration with local anesthetic for postoperative analgesia has been found to be effective in reducing pain scores, decreased need for opioids, nausea and vomiting, and increase in patient satisfaction. Perineural analgesic infusion was found to be effective in reducing opioid consumption.

Epidurals with local anesthetic combined with an opioid can provide superior analgesia than most other techniques. Epidurals are often used in high-risk surgeries with evidence showing lower pain scores than systemic opioids. Studies have also shown that epidurals may have reduced the risk of myocardial infarction and positive effects on cardiac and pulmonary outcomes. Thoracic epidural analgesia in high-risk patients has been shown to result in earlier return of bowel function and reduced risk of postoperative pulmonary complications. However, more recently some meta-analyses have reported no differences in mortality or morbidity when compared to other techniques of analgesia suggesting that giving thromboprophylaxis and adopting less invasive surgery may decrease the risk. Lower pain scores were reported with peripheral nerve analgesia than with systemic opioids; they also facilitated earlier rehabilitation and reduced the length of stay. Studies with paravertebral analgesia have shown lesser pain scores and reduced the risk of pneumonia in patients undergoing thoracotomies. There is growing interest in field blocks following truncal surgeries. Ease of insertion of regional

blocks with use of ultrasound and minimal side effects of these blocks make field blocks a suitable opioid-sparing pain management technique, especially if epidural analgesia is contraindicated or not warranted. PROSPECT (PROcedure-SPECific Pain Management) guidelines have emphasized that continuous epidural analgesia must be offered only to patients in whom the benefit outweighs the risk associated with continuous epidural analgesia and hence is not warranted in low-risk surgeries. Field blocks include and are not limited to rectus sheath block, transversus abdominus plane, erector spinae block, and quadratus lumborum block.

Intrathecal opioids provide good patient satisfaction and improved analgesia in the first 24 hours. However, the other techniques maybe preferred due to side effects such as delayed respiratory depression. The nurses need to be educated about the incidence of side effects and to recognize and treat them since the patients would be treated in wards. Subcutaneous infiltration of local anesthetics has been used for postoperative analgesia, but the analgesic efficacy is limited to the duration of analgesia of local anesthetic.

In the United Kingdom, a year-long audit to assess the rate of complications following central neuraxial blocks has been completed. The results showed that the incidence of major complications leading to permanent harm was less than previous reports. Most complications, however, were in perioperative adult patients with epidurals. Peripheral nerve catheters have increased in popularity in the past decade. They result in early mobilization, and some centers allow use on an outpatient basis facilitating early discharge and recovery. Increasing use of ultrasound for placement of nerve blocks over blind technique such as nerve stimulation or surface anatomy has led to increased block success rate and decreased neurological injuries.

Q. What are the various adjuvants that can be used for controlling postoperative pain?

Ans. Originally used for treatment of seizures, gabapentin and pregabalin have also been used to treat neuropathic pain and recently postoperative pain. They bind to a subunit of voltage-gated calcium channels, thus inhibiting calcium influx and preventing release of excitatory neurotransmitters.

Meta-analyses have shown a significant reduction in pain scores and opioid consumption. Side effects include an increased risk of sedation and dizziness, but decreased vomiting and pruritus.

Dexmedetomidine, an alpha-1 agonist, used as a sedative and analgesic agent in an intensive care setting, maybe associated with reduced opioid consumption and improved analgesia.

Ketamine, an N-methyl-D-aspartic acid (NMDA) receptor blocker, prevents central sensitization and pain sensitivity, attenuates hyperalgesia due to opioid administration, and thereby may improve opioid effectiveness. Ketamine may have preventative analgesic effects and may improve analgesia in patients who are not responding to opioids. When used in subanesthetic doses for postoperative pain management, less morphine consumption and decreased pain score and side effects were reported. Psychotomimetic effects such as hallucinations are minimal or absent in most trials.

Q. What are the barriers to pain management?

Ans. An accurate pain assessment is important for optimal pain management. Self-reporting is the most appropriate way of assessing pain but is heavily dependent on the patient's understanding of pain. Assessing a patient's fears of taking analgesics and restrictions due to side effects is also important. Some studies have shown that nurses tended to doubt what patients say about their pain and overestimate the percentage of patients who over-report their pain. Most often, they thought of the pain as "imaginary" or "psychological" when pain medications did not work. Also, some studies show that there are differences between what nurses say and what they actually do. Therefore, careful assessment of pain is important since pain is a subjective experience and there can be a variation in practice. Patients may also not volunteer information about pain since they are reluctant to interrupt busy nurses to request analgesics. Under-reporting of pain may be due to the fear of side effects.

To administer analgesia, it is important to assess pain accurately, to choose the most appropriate drug and route, and to be able to evaluate its effect. Studies indicate that patients do not receive all available analgesia despite them being in pain. It may be due to knowledge deficits, attitudes, or barriers including other priorities affecting effective pain management. Knowledge deficits regarding pharmacology, and risk and likelihood of side effects and addiction is common. Reluctance to administer opioids based on negative attitudes including fear of addiction or side effects often leads to ineffective pain management. Also, fear of side effects and addiction may be reinforced by staff reluctance.

Managing postoperative pain can be difficult with busy hospital wards, low staff numbers, limited time, inappropriate attitudes, and inadequate knowledge which impede optimal postoperative pain management. This may be mitigated by producing an environment where pain management is considered a priority by introducing regular and accurate pain assessment, a multimodal treatment approach, and a focus on responding to individual patient's needs.

There is varying evidence surrounding the benefits of preoperative education. It may increase patient satisfaction and participation. It is also part of quality of care to ensure that patients received the information they need to make decisions and to participate in their recovery. Therefore, patient education may be considered as an important part of perioperative care. Pain assessment can be more complex, in patients with dementia and learning difficulties, in elderly population, and in patients on long-term opioids for chronic pain or for recreational use.

Q. What is special caution in elderly patients?

Ans. The existence of coexisting diseases and complex medication regimes may contraindicate some types of analgesics. Age-related pharmacodynamic and pharmacokinetic changes may affect analgesic choices. Fear of giving opioids due to respiratory depression in patients is seen.

Cognitive impairment may prevent the elderly patients from using PCA but the misconception that they cannot cope with PCA may be assumed by staff in more patients than is actually the case. There is a need for simple assessment tools such as a verbal rating scale, available in large print, and objective assessment tools for patients with severe cognitive impairment. Careful choice of treatments including a multimodal approach and careful titration of opioids with regular evaluation are required.

Q. How will you manage opioid-dependent patients?

Ans. Patients who are opioid dependent require a multidisciplinary approach with appropriate teams such as drug-dependency units, general practitioners, and pain teams. Current recommendations include the use of local anesthetics, multimodal analgesia, and appropriate doses of opioids to provide analgesia and to prevent withdrawal. It is necessary to aim to match the baseline opioid requirement through either continuation of their current regime with additional doses to treat the surgical pain or using a background infusion on PCA with higher bolus doses. Patients may present with fears that staff may withhold analgesia or may wish to avoid opioid use in the fear of relapsing; therefore, communication with the patient is essential.

Predicting postoperative pain: Studies have focused on identification of predictors of pain so that early intervention may improve the pain management. Preoperative pain, anxiety, young age, obesity, surgical fear, and type of surgery (abdominal, thoracic surgery, long duration) have all been identified as strong predictors. Preoperative depression is also thought to be associated with increased pain.

Q. Describe the latest advances in pain management.

Ans. Analgesic drugs currently used for pain management often cause side effects, therefore driving the search for

new drugs or options which are more efficacious and with fewer side effects, especially around new drugs which act on peripheral opioid receptors but do not cross the blood-brain barrier and therefore have less central adverse effects.

Sustained release or extended release formulations of conventional drugs prolong the duration of analgesia to longer than 24 hours. Extended release local anesthetics have the local anesthetic drug encapsulated in biodegradable forms such as liposomes, lipospheres, and polyglycolic acid microspheres. They prolong the duration of action of local anesthetics. Extended release formulation of epidural morphine which is enclosed in microscopic lipid-based particles provides postoperative pain relief for 48 hours after a single dose. Targinact is a combination of oxycodone and naloxone which has recently been launched. The addition of naloxone blocks the action of opioid receptors in the gastrointestinal system, therefore reducing constipation, but does not reduce the analgesic effectiveness due to its extensive hepatic first-pass metabolism. Methylnaltrexone bromide and alvimopan are peripherally acting μ -opioid antagonists, which reverse some of the side effects of opioid drugs such as postoperative ileus without affecting analgesia. Iontophoretic transdermal delivery of fentanyl uses low-intensity direct current to allow rapid transfer of fentanyl from patch into skin and local circulation.

Q. How do you ensure that the patient has adequate postoperative pain relief?

Ans. The ability for improving pain management and ensuring that pain is well controlled is a shared responsibility. Patients should be encouraged to understand that they have a responsibility to inform health practitioners about their pain. Medical professionals should understand the difficulty of patients to communicate about their pain and having an understanding of this can encourage patients. Nurses should take responsibility to assess pain regularly and respond by giving appropriate treatments and reviewing its effects. Surgical teams should fully and accurately assess the patient's pain to ensure that no complications have arisen postoperatively and to ensure that pain is being treated adequately. Anesthetists are required to treat pain intraoperatively and ensure that adequate postoperative pain relief is prescribed. The APS team should ensure that education, guidelines, support, and advice are provided for patients and staff with appropriate communication, collaboration, and cooperation between healthcare professionals to optimize pain management.

The American Pain Society Guidelines on the Management of Postoperative Pain in 2016 have made 32 recommendations on safe and effective postoperative pain management on the basis of a plan of care tailored to the individual, the surgical procedure involved, and multimodal

regimens that are recommended in many situations. Out of the 32 recommendations made, 4 were assessed as being supported by high-quality evidence. They include the following:

1. The clinicians should offer multimodal analgesia, or the use of a variety of analgesic medications and techniques combined with nonpharmacological interventions, for the treatment of postoperative pain in children and adults.
2. The clinicians should provide adults and children with acetaminophen and/or NSAIDs as part of multimodal analgesia for management of postoperative pain in patients without contraindication.
3. The clinicians should consider surgical site-specific peripheral regional anesthetic techniques in adults and children for procedures with evidence indicating efficacy.
4. The clinicians should offer neuraxial analgesia for major thoracic and abdominal procedures, particularly in patients at risk for cardiac complications, pulmonary complications, or prolonged ileus.

SUGGESTED READING

1. Apfelbaum JL, Chen C, Mehta SS, Gan TJ. Postoperative pain experience: results from a national survey suggest postoperative pain continues to be undermanaged. *Anesth Analg.* 2003;97:534-40.
2. Block BM, Lie SS, Rowlingson AJ, Cowan AR, Cowan Jr JA, Wu CL. Efficacy of postoperative epidural analgesia: a meta-analysis. *J Am Med Assoc.* 2003; 290:2455-63.
3. Chou R, Gordon DB, de Leon-Casasola OA, Rosenberg JM, Bickler S, Brennan T, et al. Management of postoperative pain: a clinical practice guideline from the American pain society, the American Society of Regional Anesthesia and Pain Medicine, and the American Society of Anesthesiologists' committee on regional anesthesia, executive committee, and administrative council. *J Pain.* 2016;17(2):131-57.
4. Elia N, Lysakowski C, Tramer MR. Does multimodal analgesia with acetaminophen, nonsteroidal anti-inflammatory drugs or selective cyclo-oxygenase 2 inhibitors and patient-controlled analgesia morphine offer advantages over morphine alone? Meta-analyses of randomized trials. *Anesthesiology.* 2005;103:1296-304.
5. Liu SS, Richman JM, Thirlby RC, Wu CL. Efficacy of continuous wound catheters delivering local anaesthetic for postoperative analgesia: a quantitative and qualitative systematic review of randomized controlled trials. *J Am Coll Surg.* 2006;203:914-32.
6. Macrae WA. Chronic pain after surgery. *Br J Anaesth.* 2001;87:88-98.
7. NHS Quality Improvement Scotland. (2004). Best practice statement, postoperative pain management. [online] Available from www.nhshealthquality.org. [Last accessed February, 2020]
8. Richman JM, Lie SS, Courpas G, Wong R, Rowlingson AJ, McGready J, et al. Does continuous peripheral nerve block

- provide superior pain control to opioids? A meta-analysis. *Anesth Analg*. 2006;102:248-57.
9. Romsing J, Moiniche S, Dahl JB. Rectal and parenteral paracetamol and paracetamol in combination with NSAIDs for postoperative analgesia. *Br J Anaesth*. 2002;88:215-26.
 10. Svensson I, Sjostrom B, Halhamae H. Assessment of pain experiences after elective surgery. *J Pain Symptom Manage*. 2000;20:193-201.
 11. Toms L, McQuay HJ, Derry S, Moore RA. Single dose oral paracetamol (acetaminophen) for postoperative pain in adults. *Cochrane Database Syst Rev*. 2008;4:CD004602.
 12. Viscusi ER. Patient-controlled drug delivery for acute postoperative pain management: a review of current and emerging technologies. *Reg Anesth Pain Med*. 2008;33:146-58.
 13. Vondrackova D, Leyendecker P, Meissner W, Hopp M, Szombati I, Hermanns K, et al. Analgesic efficacy and safety of oxycodone in combination with naloxone as prolonged release tablets in patients with moderate to severe chronic pain. *J Pain*. 2008;9(12):1144-54.
 14. Yuan CS. Clinical status of methylnaltrexone, a new agent to prevent and manage opioid-induced side effects. *J Support Oncol*. 2004;2:111-7.

Pharmacokinetic Principles in Anesthesia

Vandana Agarwal, Atul Prabhakar Kulkarni

■ INTRODUCTION

Anesthesia principally consists of administration of drugs to produce desired pharmacodynamic endpoints which in turn depend on the dose of the drug administered and the pharmacokinetics of the drug. Pharmacokinetics is a series of processes, which determine the concentration of the drug both in blood and at the effect site at any time.

■ PHARMACOKINETICS

Pharmacokinetics was originally defined as the quantitative study and mathematical analysis of drug and its metabolite in the body. Simplistically, it means what body does to the drug, which includes processes such as drug absorption, distribution, metabolism, and excretion in numerical terms. It is the change in the concentration of drug over time. The knowledge of pharmacokinetics is fundamental for every anesthetist as the pharmacokinetics constants help in determining the loading dose of the drug, rate of infusion to maintain the steady-state concentration in plasma, predict the rate and extent of drug elimination, and predict modification of drug dosage in hepatic and renal impairment, which in turn help in tailoring the anesthetic drug to a patient's requirements.

In this chapter, we discuss the following pharmacokinetic variables and principles with examples where appropriate and their practical application in the practice of anesthesia:

- Volume of distribution
- Compartment models
- Half-life
- Steady state [context-sensitive half-time (CST)]
- Clearance
- First-order kinetics
- Zero-order kinetics.

■ PHARMACODYNAMICS

Pharmacodynamics describes the actions produced by the drug on the body. Thus, the effects of any drug result from a

combination of its pharmacokinetic and pharmacodynamic characteristics in an individual.

Pharmacokinetic Pathway

The passage of a drug through the body occurs through three distinct phases: uptake, distribution, and elimination.

Uptake

The rate of drug uptake and the amount of drug delivered to the target or effect site depend on the route of administration. Intravenous (IV) administration of a drug results in the entire dose entering the plasma immediately; it then passes through the pulmonary circulation into systemic circulation. Some drugs have significant uptake by the lungs, e.g., fentanyl.

Enteral administration requires the drug to cross the intestinal wall. The rate of absorption depends on surface area, pH, and, in some drugs, active systems. In general, unionized drugs (e.g., ethanol) are well absorbed throughout the intestine; absorption of weak acids (e.g., aspirin) is facilitated by a low pH and that of weak bases (e.g., morphine) by a high pH. For drugs that remain completely ionized throughout the gut (e.g., glycopyrrolate), passive gastrointestinal (GI) absorption is negligible. Even after GI absorption, a drug may not reach the systemic circulation due to the following reasons:

- Metabolism can occur in the gut mucosa (e.g., dopamine).
- First-pass metabolism in the liver via the portal vein (e.g., propranolol).
- The degree to which an administered drug reaches the systemic bloodstream is termed as its bioavailability. Bioavailability of the drug administered intravenously is 100% as shown in **Figure 1**.

Uptake after intramuscular or subcutaneous administration is primarily dependent on local blood flow rather than ionization or lipid solubility. The transdermal route is used for highly lipid-soluble drugs (e.g., nitroglycerine, fentanyl, etc.), where slow absorption eventually produces sustained blood concentrations.

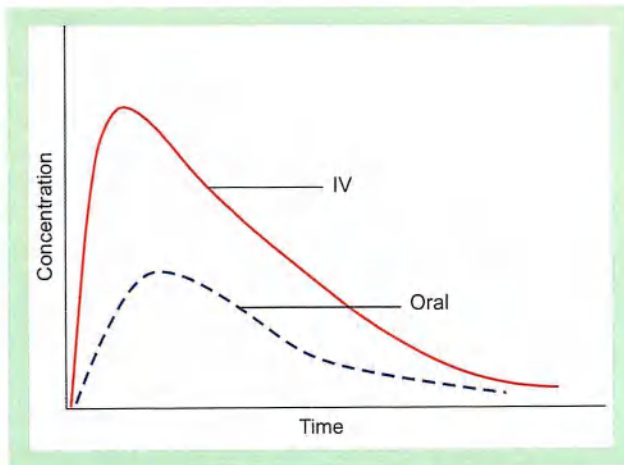


Fig. 1: Oral bioavailability. Same dose administered intravenously and orally on separate occasions. Oral bioavailability = Area under curve oral/Area under curve intravenous (IV).

Inhaled anesthetics have a different pattern of uptake. The inhaled anesthetic crosses the alveolar membrane into the blood along its partial pressure gradient. This produces an exponential wash-in, until equilibrium (i.e., the partial pressure in blood equals that in the inspired and expired gas), when no further uptake occurs. A desired clinical effect requires adequate partial pressure in the tissues, and this is achieved rapidly by:

- High fractional concentration of the inhaled drug
- High minute ventilation
- Low blood gas partition coefficient, i.e., a smaller number of molecules are needed in solution to exert a partial pressure
- A low cardiac output can also accelerate the uptake. This occurs as the circulating volume is contracted with reduced perfusion to areas outside the vessel-rich group resulting in decreased dosage of drug needed to be taken up from the alveoli.

Distribution

Following uptake, based on the characteristics of the drug, it is distributed to various tissues.

Volume of distribution (V_d): It is the apparent, i.e., not real, volume in which the drug is distributed to give a particular concentration. In practice, we measure the concentration of the drug in the plasma. It is an estimate of the extent of the distribution of the drug through body fluids and uptake by tissues. If the volume of distribution is more than the plasma volume, one can safely assume that the drug has left the intravascular or plasma compartment.

$$V_d = \text{Dose} / \text{concentration}$$

The lower the plasma concentration, the higher the volume of distribution. For example, if the amount of

drug administered is 10 mg to result in a concentration of 2 mg/L, then the volume of distribution should be 5 L.

If a drug has V_d around 5 L, one can assume that the drug remains in the intravascular compartment. These drugs are highly protein bound and as a result the plasma proteins bind to the drug and retain it in the plasma compartment. For example, warfarin and phenytoin.

If the V_d corresponds to extracellular fluid volume (around 20 L), one can assume that the drug is probably lipid insoluble and hence cannot cross the tissue cell membrane. For example, muscle relaxants.

If the V_d corresponds to total body water of 42 L, one can assume that the drug is lipid soluble and hence is able to cross the cell membrane to be distributed in the intracellular water, for example, digoxin, fentanyl, and bupivacaine.

If the V_d is more than the total body water, then presumably the drug is concentrated in certain tissues, e.g., fat and anesthetic agents. In this condition, the greater the affinity of the tissue for the drug, the more drug will be absorbed by the tissue resulting in a low concentration of the drug in the plasma. This will reflect as a very large volume of distribution. For example, propofol.

Volume of distribution at a steady state (V_{dss}): We can maintain a steady plasma concentration (C_{pss}) if we continuously replace the amount of drug eliminated from the body every minute, i.e., input = output.

$$V_{dss} = \text{Total amount of drug in the body (not the dose)} / C_{pss}$$

Pharmacological Compartments

Drugs are not distributed uniformly throughout the body. The rapidity with which a drug reaches a particular tissue is largely dependent on local blood flow, and for the purpose of analysis, similar tissue types are often grouped together into various "compartments" depending on their blood supply. The capacity of each compartment to act as a reservoir for the drug is determined by a combination of its size and affinity for the drug.

It is important to note that pharmacokinetic compartments are mathematical models and do not correspond to actual tissues; they aid in predicting the pharmacokinetic behavior of drugs.

When performing mathematical modeling, it is likely that a lipid-soluble drug that is widely distributed is likely to have several compartments; a highly ionized drug that remains in the extracellular space is likely to be best described by assuming a one-compartment model. An example of a three-compartment model is shown in **Figures 4A and B** later in the text; these correspond to vessel-rich, intermediate, and vessel-poor tissues, with a central compartment (blood), through which drugs must pass during uptake or elimination.

The movement of the drug between compartments is dependent on the concentration difference between them; as the process is exponential, the rate of transfer to the vessel-poor tissues decreases as they accumulate more drug.

Compartment Models

It is essential to understand some terminologies to make it easier to comprehend the compartment models. The following may be used in the equations of various compartment models.

e is a constant which represents mathematical interpretation of exponential changes in which the rate of change in a variable is proportional to its magnitude. Absorption, distribution, and elimination of drugs result in exponential changes in drug concentration with time.

For exponential growth, i.e., increase in drug concentration, the equation is $X = X_0 e^{kt}$ and for exponential disappearance, i.e., decrease in drug concentration, the equation is $X = X_0 e^{-kt}$, where X is the concentration at any time t , X_0 is the initial value of X at zero time, e is the base of natural logarithms (2.718), and k is a constant.

The constants k_{12} , k_{21} , k_{13} , and k_{31} represent the transfer of drug between the central and the peripheral compartments (1, 2, 3), and k_{10} is the elimination rate constant.

One-compartment model (Fig. 2): This model assumes that the body behaves like one volume and there is one clearance.

The concentration of the drug in the plasma at any one moment may be calculated by the following equation:

$$X = c * e^{-kt}$$

where X is the plasma concentration at the time t , X_0 is the initial plasma concentration or concentration at time zero, and k is the elimination rate constant and it is negative because the concentration is falling.

It is simple to understand; however, it has theoretical and practical limitations and is not directly applicable to most IV anesthetic agents.

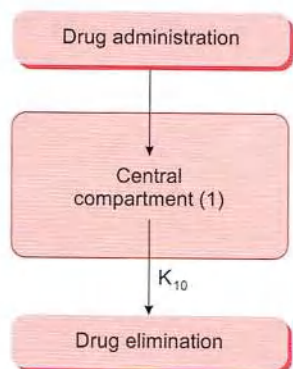
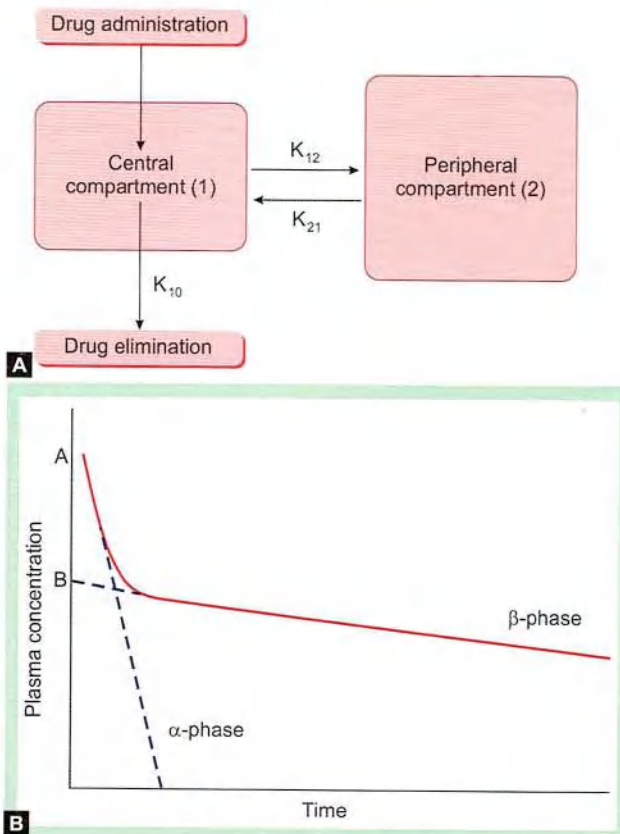


Fig. 2: One-compartment model.

Two-compartment model (Figs. 3A and B): After the initial IV bolus injection, there is a biphasic decline in plasma concentration. This initial decline is due to distribution or rapid disposition of the drug. After the distribution, equilibrium is established between the two compartments. This phase is followed by a slower decline in plasma concentration, which is due to elimination or terminal or slow disposition from the central compartment and is dependent on the rate constant k_{10} .

The plasma concentration C_p at time t can be expressed by the equation:

$\chi = Ae^{-\alpha t} + Be^{-\beta t}$ (where α and β are the slopes of α phase and β phase, respectively, i.e., elimination rate constant). Both α phase and β phase have characteristic slopes and half-lives $t_{1/2\alpha}$ and $t_{1/2\beta}$. The intercepts on the y -axis (A and B) represent the amount each half-life contributes to the decrease in plasma concentration after the initial IV dose. The two-compartment model consists of a small



Figs. 3A and B: Compartment model and its biphasic decline.

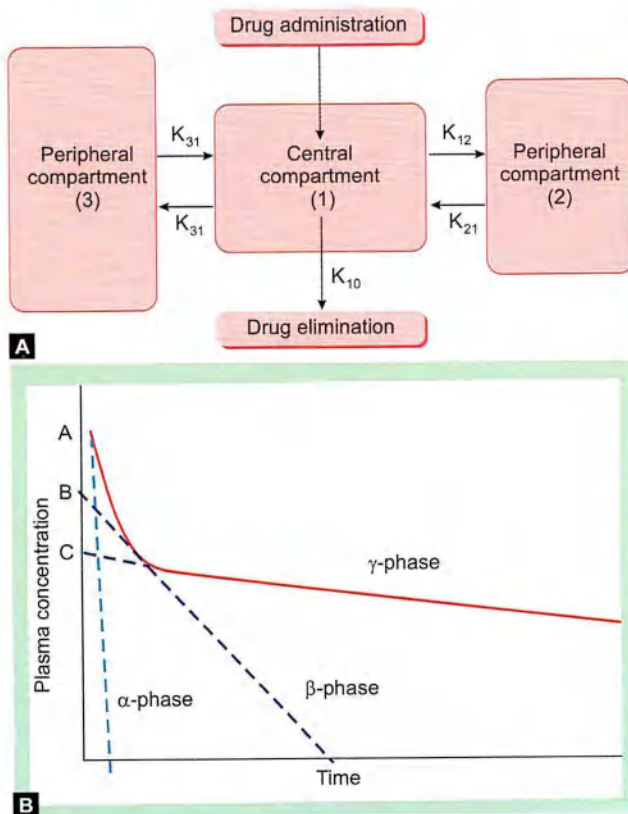
central compartment into which the drug is administered and eliminated and a larger peripheral compartment. The rate of drug transfer between compartments is governed by constants' elimination, i.e., it is governed by the rate constants k_{12} and k_{21} and the rate of elimination is governed by the rate constant k_{10} . The central compartment in case

of the two-compartment model consists of extracellular fluid and some intracellular fluid of highly or well-perfused organs and the peripheral compartment consists of less perfused tissues such as skeletal muscle and fat. An example of the drug which follows a two-compartment model intravenously is lidocaine.

Three-compartment model: The three-compartment model consists of a small central compartment into which the drug is administered and two peripheral compartments. One of the peripheral compartments is relatively small and represents well-perfused tissues and reflects the initial fall in plasma concentration (α phase) and the other is relatively large and represents less-perfused tissues and corresponds to the slow decline in plasma concentration (β phase). The rate of drug transfer between the compartments depends on the respective rate constants (k_{12} , k_{21} and k_{13} , k_{31}) and the rate of elimination is governed by the rate constant k_{10} .

There are three parts of the graph in **Figure 4**: A—the initial steep fall in the plasma concentration (C_p).

This phase is explained by the uptake of the drugs by richly perfused organs—heart and brain.



Figs. 4A and B: Three-compartment model and its triexponential decline.

B—the sustained slow fall in the C_p . This is explained by two processes that are occurring simultaneously.

The C_p continues to fall as the drug is taken up by immediately perfused organs, e.g., the muscle.

The brain and heart are fully saturated with the drug and do not take up any more drugs from the plasma. The C_p now falls, though slowly (the curve is less steep), as the muscle takes up the drug. As the C_p decreases relative to the brain and the heart concentration, the drug returns to the plasma from the brain and the heart, which tends to maintain the C_p and reduce the steepness of the curve.

C—this part is almost a straight line. This reflects that the drug is eliminated from the body at a steady rate because of a constant half-life. The rate of fall in C_p is influenced by:

- Metabolism and elimination of the drug
- The return of the drug into the plasma from the tissues tends to reduce the fall in C_p . As the drug is eliminated, the C_p falls below the levels in the tissues and the drug returns from the tissues due to a concentration gradient. The plasma concentration C_p at time t can be expressed by the equation:

$$X = Ae^{-\alpha t} + Be^{-\beta t} + Ce^{-\gamma t}$$

where α , β , and γ are the slopes of the α phase, β phase, and γ phase, respectively, i.e., elimination rate constant. The α phase, β phase, and γ phase have characteristic slopes and half-lives $t_{1/2\alpha}$, $t_{1/2\beta}$, and $t_{1/2\gamma}$, respectively.

Example: IV bolus dose of propofol.

Elimination

Exponential process: Pharmacokinetic processes occur at a rate proportional to the concentration gradient at the time. As the process continues, the concentration gradient falls, thus progressively slowing the rate of change. This results in an exponential relationship between concentration and time and applies to most drug elimination and transfer between tissues. There are two ways in which an exponential function can be described (**Fig. 5**). If a specified time period is set, the decline is defined as the fraction by which the concentration has been reduced during this interval. This is the elimination rate constant (k), expressed as time^{-1} . Alternatively, a fractional reduction in concentration is set, and the time taken to achieve this level is found.

For example, for a 50% reduction in concentration, the time taken is the half-life ($t_{1/2}$) or terminal half-life or plasma half-life. It is the time taken for the plasma concentration to decrease by 50% during the terminal phase of decline. This will be constant irrespective of the starting concentration of the drug.

$$t_{1/2} \propto \frac{V_d}{CL} = k \times \frac{V_d}{CL}$$

where V_d is the volume of distribution, CL is the clearance, and k is the constant. $t_{1/2}$ depends on the V_d and CL . A prolonged elimination half-life indicates a larger V_d or reduced clearance or both. A shorter half-life indicates an increased clearance or low V_d or both.

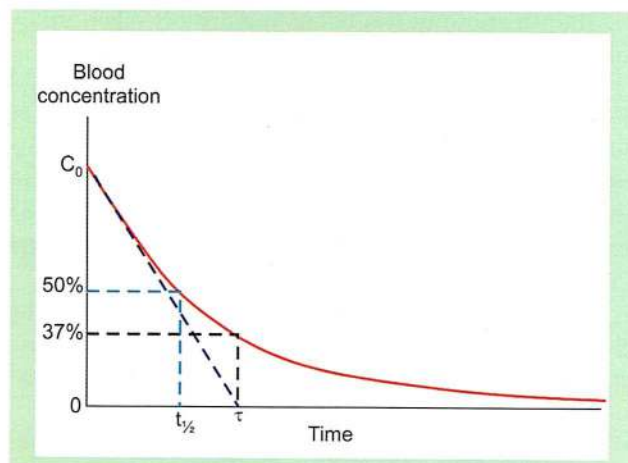


Fig. 5: Exponential decline of plasma concentration with time. (C_0 : initial concentration; $t_{1/2}$: half-life; τ : time constant).

Clinical Application of Terminal Half-life

The plasma concentration starts rising once the drug is administered. Sustained therapeutic effect is seen only when the C_p reaches a steady state. The C_{pss} is reached after 5 terminal half-lives. For example, if a drug has a half-life of 1 hour, then after the fifth dose, at the fifth hour, the plasma concentration would have reached a constant level (Figs. 6 and 7).

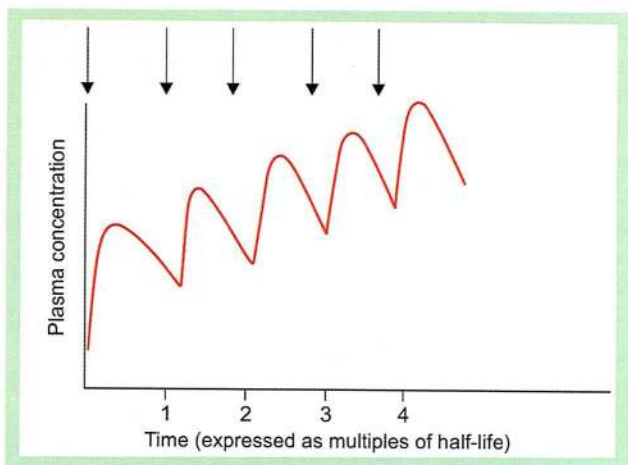


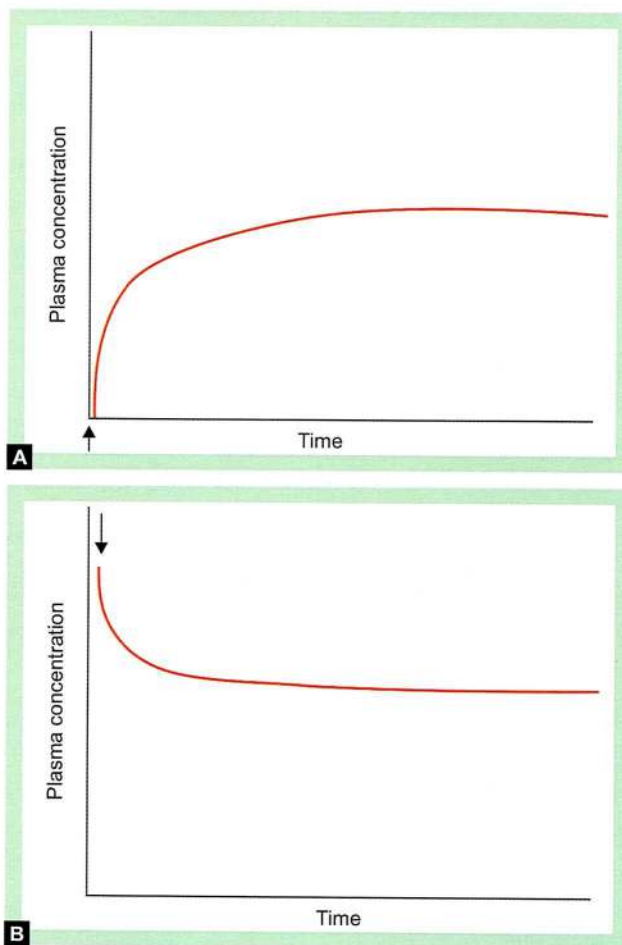
Fig. 6: Cumulation of drug when it is administered (arrows) at an interval equal to the half-life.

In general, the dosing interval is a compromise between a decline in drug concentration and the inconvenience of very frequent administration, particularly with oral medications.

For anesthetic drugs such as muscle relaxants, there is an approximate relationship between the half-life and the duration of action; e.g., suxamethonium has a half-life of 3–5 minutes and is a very short-acting muscle relaxant whereas rocuronium has a plasma half-life of 70–140 minutes and is an intermediate-acting muscle relaxant.

The relationship between terminal half-life and duration of action may not be present with other drugs. For example:

- Drugs that cross the blood–brain barrier for their clinical effect, e.g., opioids. Morphine has a shorter terminal half-life compared to pethidine but has a longer duration of action.
- Drugs which have their effect terminated by redistribution rather than elimination, e.g., IV barbiturates, propofol, fentanyl, and alfentanil.



Figs. 7A and B: (A) Cumulation of drug following IV infusion (arrow represents drug administration). (B) Cumulation of drug after an IV bolus followed by IV infusion (arrow represents drug administration).

The latent period before a steady state can be avoided by administration of loading dose equal to twice the normal dose for oral drugs. Similarly, when drugs are administered intravenously as continuous infusion, a steady state will be reached only after 4–5 terminal half-lives. This steady state can be reached earlier by administration of an IV bolus followed by continuous infusion.

Another time period that can be used to describe the curve is the time constant (τ). It is defined as the time taken to complete the elimination process if the initial rate

of decline in concentration is continued. If the initial rate of decline continued, then the plasma would be cleared of 63% of the drug in one time constant. Alternatively, the plasma concentration would be 37% of the original value at the end of one time constant.

Context-sensitive Half-time

When drugs are given by infusion, their terminal half-lives are inaccurate indicators of drug disposition and duration of

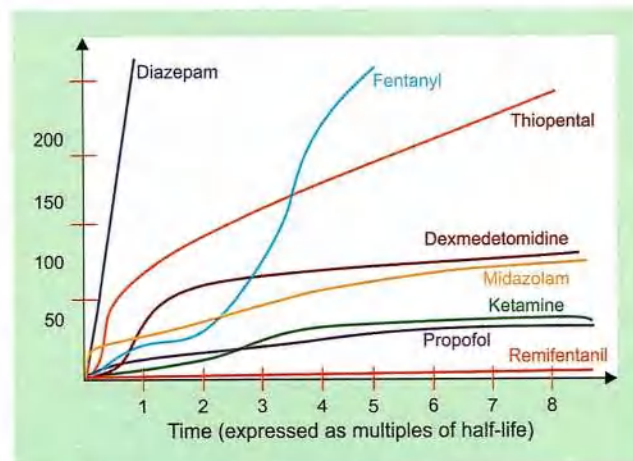


Fig. 8: Context-sensitive half-times of various anesthesia drugs.

action. In these situations, CST is used. It is defined as the time required to decrease the plasma concentration by 50% at the end of an infusion designed to maintain steady-state concentration. The context here is the duration of infusion to maintain steady-state concentration. It provides a useful guide to dosage intervals for long-term drug maintenance.

For drugs such as fentanyl in which redistribution is the main mechanism responsible for the decline in plasma concentration after a brief infusion or bolus, the CST will initially be short (Fig. 8). As the duration of infusion continues, redistribution becomes progressively less important and the CST increases, until ultimately it equals the elimination half-life. For a drug with a small volume of distribution, such as remifentanyl, redistribution is very limited and the CST changes little even with prolonged infusion.

The amount of drug cleared from the body is given by the equation:

$$O = C_p \times CL$$

where O is the amount of drug eliminated from the body every minute. If we infuse the same amount of the drug cleared from the body every minute, then the C_p would be maintained constant, i.e., at a steady state (C_{pss})

$$I = (O) = C_{pss} \times CL$$

$$C_{pss} = I/CL$$

where I is the amount of the drug infused every minute.

At a steady state, the relationship between the drug concentration and the rate of drug delivery is independent of V_d because it is governed only by total body clearance.

Rearranging the above equation,

Rate of infusion = Concentration at steady state $\times$ Clearance

To summarize, the loading dose is determined by the volume of distribution and the maintenance dose by the clearance CL of the drug.

Nonlinear Pharmacokinetics

When drug behavior is analyzed by pharmacokinetics models, it is assumed that the distribution and elimination are first-order process or kinetics.

First-order kinetics implies that the rate of transfer of drug from one compartment to another is proportional to the drug concentration, i.e., the amount of drug being removed is a constant fraction in unit time rather than a constant amount. This is not always the case, as drug distribution and elimination maybe dependent on carrier transport and can get saturated. Similarly, many reactions with drug metabolism are saturable and proceed at a maximal rate if the drug concentration is high. Once the relevant blood concentration is reached, elimination becomes constant, limited to a maximum amount in unit time. In these conditions, the drug transport and metabolism occur at a high but a constant rate irrespective of the concentration of the drug, i.e., a constant proportion of the drug is cleared. This is termed *nonlinear pharmacokinetics* or *zero-order kinetics* and occurs with capacity-limited elimination of drugs.

This is not a common phenomenon; however, it can occur in dangerously high concentrations with continued, unmonitored drug administration or prolonged infusion, e.g., ethanol, salicylates, and phenytoin. The elimination of drugs occurs at a constant rate irrespective of the drug concentration and when the concentration decreases to a certain level, i.e., the systems are not saturated any more, then the kinetics changes from zero order to first order.

Clearance

Clearance (CL) is defined as the volume of blood or plasma from which the drug is completely cleared in unit time (mL/min). It is also defined as the rate of drug elimination (mg/min) per unit of blood or plasma concentration (mg/mL). A drug can undergo clearance by the kidneys, liver, or other tissues. Total clearance is the sum of different ways of drug elimination through various organs in the body.

$$CL = CL_R + CL_H + CL_X$$

where CL_R is renal clearance, CL_H is hepatic clearance, and CL_X is clearance by other routes.

The clearance of a drug through an organ (renal or hepatic) depends on:

- Extraction ratio (ER)
- Organ plasma flow (Q)
- $CL = ER \times Q$

Renal clearance can be directly measured or calculated by:

$$CL_R = \text{Dose of drug in the urine/AUC}$$

where AUC is the area under the plasma concentration-time curve during the elimination phase. If the ratio of renal clearance to total clearance of the drug is >70%, then the drug is predominantly eliminated by kidneys from the body. It implies that the drug will cumulate in renal failure, whereas if the CL_R is <30% renal impairment has little or no impact on elimination.

Hepatic Clearance

ER is an overall ability of the liver to remove a drug from the hepatic capillaries and reflects drug metabolism and biliary secretion.

$$ER = \frac{C_a - C_v}{C_a} = 1 - \frac{C_v}{C_a}$$

where C_a is the concentration of drug in the mixed portal venous and hepatic arterial blood and C_v is the drug concentration in the hepatic venous blood. Elimination of the drugs by liver is dependent on:

- Hepatic blood flow
- Proportion of unbound drug in the plasma
- Activity of the hepatic enzymes.

$$CL_H = ER \times Q$$

It is also expressed in terms of blood flow and intrinsic clearance. Intrinsic clearance is the maximal ability of the liver to irreversibly eliminate drugs by metabolism or biliary excretion and is independent of blood flow:

$$CL_H = Q \times \frac{(CL_i \times f)}{Q + (CL_i \times f)} \quad \text{Equation 1}$$

where CL_i is the intrinsic clearance, which represents the rate at which liver water is cleared of the drug (mL/min) and f is the fraction of unbound drug in plasma.

When the intrinsic clearance is relatively low compared to the blood flow, then

$$Q + (CL_i \times f) = Q \quad \text{Substituting in Equation 1}$$

$$CL_H = Q \times \frac{(CL_i \times f)}{Q} = CL_i$$

In these circumstances, hepatic clearance is dependent on the intrinsic clearance and the unbound fraction of the drug in plasma. This is termed capacity-limited elimination, e.g., phenytoin, warfarin, barbiturates, and benzodiazepines. These drugs have limited first pass effect after oral administration and low hepatic extraction ratio. Their clearance is relatively low and will

be unaffected by changes in hepatic blood flow; however, it will be profoundly influenced by the activity of the hepatic enzymes. The unbound fraction of the drug can have an effect as well, particularly for drugs with a high degree of protein binding.

When the intrinsic clearance is relatively large compared to the blood flow, then

$$Q + (CL_i \times f) = (CL_i \times f) \quad [\text{substituting in Equation (1)}]$$

$$CL_H = Q \times \frac{(CL_i \times f)}{(CL_i \times f)} = Q$$

In these conditions, hepatic clearance is primarily dependent on hepatic blood flow, and this is termed flow-limited elimination, e.g., opioids analgesics, lidocaine, and tricyclic antidepressants. The half-life can be modified by alterations in hepatic blood flow, but is not affected by the hepatic enzymatic activity or protein binding. The hepatic extraction is high and there is substantial first-pass effect for orally administered drugs.

PRACTICAL APPLICATIONS

Total Intravenous Anesthesia and Target-Controlled Infusions

Understanding of pharmacokinetics is fundamental for safe practice of total intravenous anesthesia (TIVA). Misunderstanding or misconceptions can cause accidental awareness as found in the NAP5 (National Audit Project, UK). TIVA requires maintenance of clinically appropriate plasma concentration. This is best achieved by target TCI.

Target-controlled infusions work on the principle of bolus/elimination/transfer (BET). The initial drug bolus loads the central compartment and the infusion rate takes into consideration the intercompartmental transfer and elimination. Once the target concentration is dialed, the infusion pump delivers a bolus and infusion is continued till the target concentration is achieved. The drug diffuses from plasma into the brain and equilibration is achieved within 4–5 half-times. If the target plasma concentration is increased, the infusor delivers an additional bolus and the infusion rate is increased to match additional transfer into the central compartment and elimination at a higher rate. When the target plasma concentration is decreased, then the infusion stops for a period until new target concentration is achieved and starts at a lower rate. Calculations are repeated every 10 seconds and the infusion rate adjusted until target plasma concentration is achieved.

Establishing a Steady State

- If a drug is given as a constant infusion, it will eventually reach a steady state (i.e., with the whole V_{dss} containing the drug at a stable concentration, and elimination

occurring at the same rate as administration). It takes 4–5 elimination half-lives to achieve this steady state.

- The target concentration can be attained far more rapidly using an initial loading dose followed by further additional drug in a declining exponential fashion as redistribution to other tissues occurs.

Predictability

- The pharmacokinetic behavior of inhalational anesthetic agents is far more predictable than IV anesthetic agents because of the physics of gas/vapor solution in liquid. At equilibrium, the partial pressure of an inhalational agent in blood (and other tissues) will precisely equal that in the inhaled gas mixture. Furthermore, the end-tidal partial pressure of an inhalational agent can be measured in real time, providing a value very close to that in arterial blood.
- Established pharmacokinetic and pharmacodynamic data are derived from averaged population studies. One must accept individual variation in both pharmacokinetics and pharmacodynamics even with most sophisticated dosage schemes for IV drugs.

During the conduct of anesthesia, the above variation should be taken into consideration along with patients' clinical condition and modify target (or effect) site concentration/dosages for TCI/TIVA, respectively, to avoid adverse hemodynamic effects.

Practical Aspects of Target-Controlled Infusions

- Dedicated IV line for TCI–TIVA with an antisiphon valve.
- Choose the model for propofol suitable for the patient.
- Input demographic variables.
- Selecting TCI targets should take into consideration administration of concomitant opioids and premedication.
- Assess the level of consciousness before administering a relaxant.
- Muscle relaxant should not be administered before lack of response to jaw thrust.
- Use processed electroencephalography (EEG), especially when muscle relaxants are used.
- Adjust target C_p or target effect site concentration (C_{et}) as per surgical stimulus.
- Targets should not be reduced inappropriately just to promote a more rapid recovery.
- Infusions are more safely stopped once the final sutures are applied.

Caution with Target-Controlled Infusions

- Training and education regarding the pharmacokinetic aspect of PK models is very essential for smooth conduct of anesthesia.

- Familiarize yourself and the anesthesia team with the TCI equipment, PK model, and pitfalls before beginning the case.
- A dedicated IV line is essential for administering propofol, and this line should always be visible during the anesthetic as unrecognized accidental disconnection can lead to awareness.
- Set low-pressure and high-pressure alarms for detecting disconnection and tissue extravasation.
- Antireflux valve should be used for IV fluid to prevent flow back into the fluid (if the same IV is used for administering anesthesia), and an antisiphon valve should be used for TCI extension tube to prevent siphoning back into the pump.
- The next syringe should be kept ready before the previous one finishes. The pump alarms when the syringe is near empty.
- NAP5 audit conducted in the hospitals across National Health Service in the United Kingdom identified technical failure and poor application of knowledge in most cases of anesthetic awareness using TIVA. However, 75% of these cases could have been prevented by suitable education and training. NAP5 emphasized that processed EEG is necessary in patients who require neuromuscular paralysis.
- The National Institute of Health and Care Excellence (NICE) has also recommended deployment of a processed EEG device when administering TIVA (www.nice.org.uk/guidance/dg6/chapter/1-Recommendations).

SUGGESTED READING

1. Al-Rifai Z, Mulvey D. Principles of total intravenous anaesthesia: basic pharmacokinetics and model descriptions. *BJA Educ.* 2016;16(3):92-7.
2. Calvey TN, Williams NE. Principles and Practice of Pharmacology for Anaesthetists, 5th edition. New Jersey: John Wiley & Sons, Inc.; 2008.
3. Moffat A, Milne B. Pharmacokinetics in anaesthesia. *Can Anaesth Soc J.* 1983;30(3):300-7.
4. Nimmo AF, Cook TM. Total intravenous anaesthesia. 5th National Audit Project (NAP5). Accidental Awareness during General Anaesthesia in the United Kingdom and Ireland Report and Findings—Chapter 18. The Royal College of Anaesthetists and the Association of Anaesthetists of Great Britain and Ireland; 2014.
5. Pandit JJ, Andrade J, Bogod DG, Hitchman JM, Jonker WR, Lucas N, et al. 5th National Audit Project (NAP5) on accidental awareness during general anaesthesia: summary of main findings and risk factors. *Br J Anaesth.* 2014;113(4):549-59.
6. Roberts F, Freshwater-Turner D. Pharmacokinetics and anaesthesia. *Contin Educ Anaesth. Crit Care Pain.* 2007;7:25-9.
7. Schwilden H. Pharmacokinetics as related to anaesthesia. *Curr Anaesth Crit Care.* 1997;8:202-6.

Fluid Management in Major Abdominal Surgery

Vandana Agarwal, Bindiya Salunke

Q. Describe the stress response to surgery and pathophysiological effects of fluid balance.

Ans. Sixty percent of the total body weight (TBW) is made up of water, which is distributed within the extracellular (20% of TBW) and intracellular (40% of TBW) spaces. Plasma volume is maintained by the equilibrium of Starling forces, i.e., hydrostatic pressure that drives the fluid out and oncotic pressure that draws the fluid in. Plasma oncotic pressure is mainly maintained due to albumin (75%), but hemoglobin (20%) and globulins (5%) also contribute. In addition, there is exchange of fluid and electrolytes between the extracellular space and the gastrointestinal system to maintain the milieu interior.

Stress response following surgery is a combination of endocrine and inflammatory mediators. Hormonal response to surgery occurs for the preservation of adequate blood volume. This causes the secretion of antidiuretic hormone which enhances resorption of water in the distal tubules resulting in reduced postoperative diuresis and hyponatremia. Stimulation of the renin-angiotensin-aldosterone axis leads to increased retention of salt and water and excretion of potassium. This affects the distribution of fluid and causes an expansion of the extracellular fluid and thus edema along with some degree of oliguria. Therefore, oliguria in the postoperative period does not always mandate fluid replacement until there is evidence of hypovolemia, e.g., bleeding or ongoing losses from the gastrointestinal system. Arginine vasopressin, released from the posterior pituitary, acts on the kidney and promotes water retention and production of concentrated urine. Increased vasopressin secretion may continue for 3–5 days, depending on the severity of the surgical insult and the development of complications. Hormonal changes are directed to preserve the extracellular fluid and circulating blood volumes. Therefore, an appropriate prescription of postoperative fluids with right concentration and volume is essential. The ability of the kidneys to compensate

for prescription errors is limited even in the absence of acute kidney injury due to the hormonal response to injury. Surgery stimulates the adrenal cortex via the adrenocorticotrophic hormone (ACTH) leading to increased secretion of cortisol. Metabolic effects of increased cortisol secretion cause hyperglycemia because of its effect on carbohydrate, fat, and protein metabolism. In addition, it has an anti-inflammatory effect and interferes with the synthesis of inflammatory mediators, e.g., prostaglandins. This may in turn limit fluid shift.

Inflammatory mediators (cytokines) are released in response to injury and they increase the endothelial/capillary permeability and cause vasodilatation. The quantum of release is directly proportional to the magnitude of surgery and can have both local and systemic consequences.

Q. What is the goal of perioperative fluid therapy/management?

Ans. The primary goal of perioperative fluid therapy is to maintain or restore an adequate circulating volume with an adequate fluid and electrolyte solution to ensure end-organ perfusion and oxygen delivery to the tissues. Both hypovolemia and hypervolemia are associated with increased postoperative morbidity. Hypovolemia causes hypotension impaired tissue oxygenation, and inadequate organ perfusion (e.g., acute kidney injury) which can vary from organ dysfunction to organ failure which, in turn, is associated with poor postoperative outcomes. Hypervolemia causes interstitial edema, impaired tissue oxygenation, dilutional coagulopathy, and cardiopulmonary complications associated with fluid overload. Interstitial edema presents differently in different organs. In the lungs, it impairs respiratory exchange, which increases the risk for postoperative respiratory failure, pneumonia, and/or pulmonary edema). In the gastrointestinal system, it decreases motility and ileus,

increases tension at the anastomotic site, increasing the risk of anastomotic dehiscence.

Q. Briefly describe the conventional fluid management approach.

Ans. Conventionally, large volumes of intravenous (IV) fluids were used intraoperatively to achieve good blood volume. Patients were considered hypovolemic due to various reasons such as preoperative prolonged fasting [>8 hours, patients were kept nil by mouth (NBM) from midnight], bowel preparation, intraoperatively losses secondary to bleeding, third space loss due to surgical exposure, ongoing loss from perspiration and urinary output and hypotension induced by anesthetic agents and vasodilatory effect of neuraxial anesthesia. Crystalloid administration for relative hypovolemia hypotension secondary to reduced sympathetic tone following neuraxial or general anesthesia was considered harmless, and administration of vasopressors to counteract, it was considered to be associated with harm especially to the kidneys. In addition, the 4-2-1 rule for perioperative fluid therapy was also adopted as a guide to replace this fluid loss.

Patients undergoing major abdominal surgery received:

- Preoperative crystalloid loading at 2 mL/kg/h of fasting.
- Infusions of crystalloids at three to four times the actual blood loss.
- Patients were frequently administered 4–8 mL/kg/h of crystalloid based on suspected insensible losses, such as third spacing and evaporation.
- This formula may result in basal crystalloid infusion rates up to 20 mL/kg/h. The infusion rates were often titrated to obtain a urine output of 0.5–1 mL/kg/h.

Currently, with an increasing number of surgeons following enhanced recovery after surgery (ERAS) protocols, preoperative fasting is minimal such that patients are allowed to drink water throughout the night up to 2 hours before surgery, carbohydrate supplementation is given the night before and 2 hours before surgery, and bowel preparation is done in selective patients. As a consequence, these patients are not dehydrated and thirsty or hypovolemic. Also, insensible losses are considered minimal. Thus, compared to traditional practice, these patients do not receive any replacement for starvation and insensible losses and are given maintenance and replacement of blood loss or nasogastric aspirate if inserted.

Q. What are the different strategies of IV fluid administration perioperatively?

Ans. The following strategies are described for perioperative fluid management:

- Supranormal
- Liberal

- Restrictive or zero balance
- Goal-directed.

Shoemaker et al. in the 1980s observed that survivors following high-risk surgery had a higher cardiac index and oxygen delivery compared to nonsurvivors and proposed targeting supranormal physiological parameters in the perioperative setting to meet the increased postoperative metabolism. They targeted a cardiac output of >4.5 L/min/ m^2 and an oxygen delivery of >600 mL/min/ m^2 . Pulmonary artery catheters were inserted preoperatively, and fluids and inotropes were used to achieve supranormal values. They observed that it was associated with reduced complications, duration of mechanical ventilation, shorter ICU and hospital length of stay, and reduced costs compared to standard care. However, this was later criticized because of dissimilar groups and lack of randomization. Subsequent trials demonstrated no benefit but worsened postoperative outcomes.

It originated from the concept of the so-called third space loss and fear of perioperative hypovolemia. It was believed that surgical injury causes fluid shift from body compartments into nonanatomical space (third space) leading to loss of extracellular fluid. This led to administration of 15 mL/kg/h initially and decreasing subsequently as fluid algorithm. Studies with sound methodology have debunked this concept and therefore this concept should be abandoned. Postoperative weight gain following liberal fluid therapy has been demonstrated to be associated with worse perioperative outcomes. This subsequently led to the concept of restrictive fluid strategy or zero balance strategy. In abdominal surgery, restrictive strategy aimed at zero balance (weight gain <1 kg) in comparison to liberal strategy was associated with reduced complications, postoperative stay and earlier return of gastrointestinal function, thus suggesting that liberal fluid was associated with greater tissue edema and poor wound healing. However, the literature is abundant with various definitions of liberal and restrictive strategies. Lack of standardized definitions for standard, liberal, or restrictive strategies has made comparison and inferring from the available literature very difficult and confusing. All these studies have no pre-identified hemodynamic goals or targets and have varying endpoints. This subsequently led to the development of goal directed fluid therapy (GDT).

Goal directed therapy

GDT uses functional hemodynamic parameters to optimize cardiac output by administration of fluids and vasopressor/inotropes. The OPTIMISE (*OPTimisation of Peri-operative Cardiovascular Management to Improve Surgical outcome*) trial evaluated the impact of cardiac output monitoring along with fluids and dopexamine in high-risk patients

undergoing intra-abdominal surgery. GDT probably reduces the likelihood of morbidity and mortality by about 7% and up to 14%, respectively. Inclusion of this data in a systematic review with seven additional trials observed a significant reduction in complication rate, infections, and shorter postoperative stay in the GDT group. The RELIEF (Restrictive vs Liberal Fluid Therapy for Major Abdominal Surgery) trial compared restrictive (intraoperative 5 mL/kg/hr, postoperative 0.8 mL/kg/hr) vs liberal fluid strategy (intraoperative 10–8 mL/kg/hr, postoperative 1.5 mL/kg/hr) in major abdominal surgery. The authors observed an increased incidence of acute kidney injury in the restrictive group with no difference being seen in disability-free survival between two groups. The authors recommend a modestly liberal regimen over a truly restrictive regimen. A multicenter FEDORA trial optimized the patient's fluid status to maximize stroke volume and then added a vasopressor. They observed a significant reduction in postoperative complications and length of stay in the GDT group. The INPRESS (Intraoperative Norepinephrine to Control Arterial Pressure) trial, conducted in patients undergoing abdominal surgery at increased risk of postoperative complications, individualized systolic blood pressure management within 10% of baseline by administration of fluids followed by vasopressors was associated with a significant reduction in postoperative organ dysfunction. A recent systematic review of perioperative GDT with alpha-1 adrenergic agonists found significant reduction in postoperative complications, length of stay, and early functional GI recovery with no impact on mortality.

In the era of modern surgical practice with increased emphasis on minimally invasive surgery a modestly liberal strategy, which does not cause substantial fluid retention with or without vasopressors, appears safe. In summary, one has to individualize the hemodynamic targets and therapy and follow the Goldilocks principle of not too much or too little but just the right amount fluid.

Q. What are the recommendations for clear fluids preoperatively?

Ans. The American Society of Anesthesiologists guidelines allow unrestricted intake of clear fluids up to 2 hours before elective surgery. Multiple randomized controlled trials (RCTs) have proved that risk of aspiration decreases when clear liquids are allowed 2 hours before a procedure.

When any patient is kept fasting overnight, he continually produces saliva along with endogenous gastric secretions and this adds to both volume and acidity to stomach content. Allowing clear fluids 2 hours prior to surgery is beneficial due to the following reasons:

- It reduces thirst and hunger and thus decreases endogenous gastric secretions and salivation and hence

does not contribute in increasing the gastric volume preoperatively.

- The oral intake of complex (maltodextrin) carbohydrate drink up to 2 hours before surgery reduces insulin resistance, provides a superior perioperative glycemic control, and reduces the risk of nosocomial infection.

Q. Which fluids should be administered perioperatively?

Ans. Fluid therapy should be considered for maintenance and replacement of losses (volume therapy). Maintenance fluids take into consideration the insensible losses and urine output from the beginning of the preoperative fast. Usually, this should be 1–1.5 mL/kg and increased depending on the nature of surgery. It should be ideally similar to plasma, i.e., isotonic and balanced, e.g., Ringer's lactate, Hartmann's and Plasmalyte.

Studies from intensive care have questioned the role of normal saline as maintenance fluid due to adverse effects of hyperchloremic acidosis and increased incidence of acute kidney injury. Normal saline, however, will have its role in patients with hyponatremia and those with neurological dysfunction. Two studies that compared normal saline with balanced salt solution in an emergency department and critically ill patients found lower incidence of acute kidney injury in the balanced solution group. In critically ill patients, it was associated with reduced mortality and new-onset renal replacement therapy. However, neither of the trials was done in perioperative patients.

Q. Describe the clinical approach to assess the needs for IV fluids.

Ans. The clinical approach can be described by five Rs:

1. *Resuscitation* is indicated in hypovolemic patients. If there are no ongoing losses, then routine maintenance fluid should be prescribed.
2. *Replacement*: After resuscitation, if there are ongoing losses or redistribution of fluid then this should be replaced in addition to maintenance volume. For example, ileostomies, fistulae, nasogastric tube drainage and surgical drains or urinary tract (e.g., when recovering from acute kidney injury). In patients with fever or burns, there can be high insensible losses. If not replaced timely, the patient can become hypovolemic and may need resuscitation.
3. *Redistribution*: Redistribution occurs, e.g., in critically ill with sepsis, in postoperative patients, and in patients with severe renal, liver, or cardiac conditions. These patients develop peripheral or pulmonary edema, weight gain, delayed wound healing, and compartment syndromes.
4. *Routine maintenance* should be prescribed if there are no ongoing losses. An average person daily requires

25–30 mL/kg of water, 50–100 g of glucose, and approximately 1 mmol/kg of Na⁺ and K⁺. Patients who are elderly, frail, with cardiac failure, or renal impairment maintenance should be restricted to 20–25 mL/kg/day. When the patient no longer needs IV therapy, they should be switched to the enteral route as soon as possible. The enteral route is the safest, simplest, and most effective way of administering fluids. Either oral or tube feeds should be established at the earliest after surgery when it is considered surgically safe.

5. *Reassessment* should be done periodically to evaluate for ongoing losses and replacement volume should be administered. Fluids and electrolytes should be prescribed for 24 hours and modified if there are losses as and when detected.

The above-mentioned approach is a continuum of care in patients undergoing surgery that should be followed in the perioperative period.

■ SUGGESTED READING

1. Al-Ghamdi AA. Intraoperative fluid management: past and future, where is the evidence? *Saudi J Anaesth.* 2018;12(2):311–17.
2. Bamboat ZM, Bordeianou L. Perioperative fluid management. *Clin Colon Rectal Surg.* 2009;22(1):28–33.
3. Calvo-Vecino JM, Ripollés-Melchor J, Mythen MG, Casans-Francés R, Balik A, Artacho JP, et al. Effect of goal-directed haemodynamic therapy on postoperative complications in low-moderate risk surgical patients: a multicentre randomised controlled trial (FEDORA trial). *Br J Anaesth.* 2018;120(4):734–44.
4. Feng S, Yang S, Xiao W, Wang X, Yang K, Wang T. Effects of perioperative goal-directed fluid therapy combined with the application of alpha-1 adrenergic agonists on postoperative outcomes: a systematic review and meta-analysis. *BMC Anesthesiol.* 2018;18:113.
5. Futier E, Lefrant J, Guinot P, Godet T, Lorne E, Cuvillon P, et al. Effect of individualized vs standard blood pressure management strategies on postoperative organ dysfunction among high-risk patients undergoing major surgery: a randomized clinical trial. *JAMA.* 2017;318(14):1346–57.
6. National Institute for Health and Care Excellence. (2017). Intravenous fluid therapy in adults in hospital. [online] Available from <https://www.nice.org.uk/guidance/cg174>. [Last accessed February 2020]
7. Pearse RM, Harrison DA, MacDonald N, Gillies MA, Blunt M, Ackland G, et al. Effect of a perioperative, cardiac output-guided hemodynamic therapy algorithm on outcomes following major gastrointestinal surgery: a randomized clinical trial and systematic review. *JAMA.* 2014;311(21):2181–90.
8. Shoemaker WC, Appel PL, Kram HB, Waxman K, Lee TS. Prospective trial of supranormal values of survivors as therapeutic goals in high-risk surgical patients. *Chest.* 1988;94:1176–86.
9. Timothy EM. Perioperative fluid therapy for major surgery. *Anesthesiology.* 2019;130:825–32.
10. Voldby AW, Brandstrup B. Fluid therapy in the perioperative setting: a clinical review. *J Intensive Care.* 2016;4:27.

SECTION

2

Anesthesia Equipment and Table Viva

- **Arterial Blood Gas Analysis**

Vijaya Prakash Patil, Jeson Rajan Doctor, Bindiya Salunke

- **Mechanical Ventilation**

Atul Prabhakar Kulkarni, Sohan Lal Solanki, Jigeeshu Vasistha Divatia

- **Pulmonary Function Tests**

Vijaya Prakash Patil, Madhavi Desai, Debashree Lahiri

- **Anesthesia Machine**

Nayana Amin, Vandana Agarwal

- **Vaporizers**

Priya Ranganathan, Madhavi Shetmahajan

- **Anesthesia Breathing Systems**

Sheila Nainan Myatra, Parmanand Jain

- **Endotracheal Tubes, Lung Isolation Devices and Combitube**

Atul Prabhakar Kulkarni, Aparna Chatterjee, Swapnil Parab

- **Cardiopulmonary Resuscitation**

Sheila Nainan Myatra, Bhakti Trivedi, Amol Trimbakrao Kothekar

- **Airways, Connectors, Laryngoscopes and Non-rebreathing Valves**

Nayana Amin, Sudivya Sharma, Sukhada Savarkar

- **Electrocardiography**

Reshma Ambulkar, Swapnil Parab, Bindiya Salunke

- **X-ray Interpretation for the Budding Anesthesiologist**

Atul Prabhakar Kulkarni, Aparna Chatterjee, Debashree Lahiri

- **Supraglottic Airway Devices**

Vandana Agarwal, Sukhada Savarkar, Atul Prabhakar Kulkarni

- **Regional Anesthesia Instruments**

Parmanand Jain, Atul Prabhakar Kulkarni, Jeson Rajan Doctor

- **Nerve Locator and Peripheral Nerve Stimulator**

Nayana Amin, Sudivya Sharma

- **Oxygen Therapy Devices and Manual Resuscitator**

Nayana Amin, Vijaya Prakash Patil, Sohan Lal Solanki

- **Videolaryngoscopes**

Atul Prabhakar Kulkarni, Madhavi Desai, Shilpushp Jagannath Bhosale

- **Cardiopulmonary Exercise Test (CPET/CPEX/CPX)**

Vandana Agarwal, Vijaya Prakash Patil

Arterial Blood Gas Analysis

Vijaya Prakash Patil, Jeson Rajan Doctor, Bindhya Salunke

Q. Explain basic physiological processes reflected in ABG.

The arterial blood gas (ABG) provides us with rapid information on three physiologic processes:

1. **Alveolar ventilation:** The maintenance of CO_2 level reflected by arterial CO_2 tension (PaCO_2) at any given moment depends on the quantity of CO_2 produced in body and its excretion through alveolar ventilation (VA) and can be expressed by the equation, $\text{PaCO}_2 \sim \text{CO}_2/\text{VA}$. The alveolar ventilation is that portion of total ventilation that participates in gas exchange with pulmonary blood. Thus PaCO_2 is the best index for assessment of alveolar ventilation. High PaCO_2 (>45 mm Hg) indicates alveolar hypoventilation and low PaCO_2 (<35 mm Hg) implies alveolar hyperventilation.
2. **Oxygenation:** The ultimate aim of oxygenation is to provide adequate delivery of oxygen to tissues. This is a function of cardiopulmonary system and various factors like arterial oxygen tension (PaO_2), hemoglobin content and saturation with oxygen and cardiac output. The PaO_2 and SaO_2 is primarily used for assessment of oxygenation status. Approximately, 98% of oxygen is carried in blood in the combined state with hemoglobin. Hypoxemia is defined as PaO_2 of less than 80 mm Hg at sea level in an adult patient breathing room air, the similar decrease in cells/tissues is known as hypoxia. Tissue hypoxia is unlikely in mild hypoxemia ($\text{PaO}_2 = 60\text{--}79$ mm Hg) and is almost always associated with severe hypoxemia ($\text{PaO}_2 < 45$ mm Hg). PaO_2 must always be interpreted in relation to concentration of inspired oxygen FiO_2 and age. Relation between PaO_2 and FiO_2 : PaO_2 alone provides little information regarding efficiency of oxygen loading into the pulmonary capillary blood. In other words, it does not quantitate the physiological shunt, which helps in assessment of the severity of underlying disease in lungs and in guiding oxygen therapy. Since, the normal PaO_2 in an adult breathing room air with FiO_2 of 0.2 is

80 to 100 mm Hg, the normal values for $\text{PaO}_2/\text{FiO}_2$ ratio or oxygenation ratio are 400–500 mm Hg or 4.0 to 5.0 respectively. $\text{PaO}_2/\text{FiO}_2$ ratio of less than 200 most often indicates a shunt greater than 20%.

3. Acid-base balance.

Q. How does age affect oxygenation?

$$\text{PaO}_2 = 104 - (0.27 \times \text{age})$$

more crudely, normal oxygenation for age is roughly 1/3 of the patient's age subtracted from 100. Using this estimation, a 60-year-old patient should have a PaO_2 of 80.

Q. Explain terms shunt and dead space.

Shunt and dead space are terms used to describe extreme conditions where either blood flow or ventilation does not meet the other in the lung as it should for gas exchange to take place.

Shunt: It is part of the cardiac output that returns to the left heart without the benefit of exposure to ventilated alveoli.

Dead space: It is part of inspired air that is exhaled without the benefit of exposure to perfused alveoli. It is normal for a small percentage of air in the lungs not to reach blood. Air in the nasopharynx, trachea and bronchi does not reach the alveoli before exhalation:

- More than this amount of "dead space" can lead to hypoxia
- Circulatory problems that result in a shunt are called "V-Q shunt" or "V-Q mismatch" where:
 - V represents "ventilation" and
 - Q represents "perfusion". V and Q can be compared directly using a nuclear medicine procedure called a "V-Q scan" or a "lung scan."

Classic Shunt Equation

$$\text{Qs}/\text{Qt} = (\text{CcO}_2 - \text{CaO}_2)/(\text{CcO}_2 - \text{CvO}_2)$$

CcO_2 = End capillary O_2 content

CaO_2 = Arterial O_2 content

CvO_2 = Mixed venous.

V/Q Ratio

Normal V (ventilation) is 4 L of air per minute. Normal Q (perfusion) is 5 L of blood per minute:

- So normal V/Q ratio is 4/5 or 0.8.

When the V/Q is higher than 0.8, it means ventilation exceeds perfusion.

When the V/Q is < 0.8, there is a VQ mismatch caused by poor ventilation or excess perfusion as in one lung anesthesia.

Q. What is A-a gradient?

It predicts the degree of shunt by comparing the partial pressure of O₂ in the (A) alveoli to that in the (a) artery.

The difference between them gives us an idea how well the oxygen is moving from the alveoli to the arterial blood (PAO₂ - PaO₂). The PaO₂ is obtained from the ABG. The PAO₂ (partial pressure of oxygen in the alveolus) is obtained from the alveolar gas equation:

$$PAO_2 = (760 - 47) \times FiO_2 - PaCO_2 / \text{Respiratory Quotient} \\ (0.8 \text{ or } 1) \text{ OR}$$

$$PAO_2 = 150 - 1.25 (PaCO_2)$$

$$A\text{-}a \text{ gradient} = [150 - 1.25 (PaCO_2)] - PaO_2$$

A normal A-a gradient is 10–20 mm Hg.

A-a gradient when the patient is not breathing room air is calculated as:

$$A\text{-}a \text{ gradient} = \{(FiO_2)(760 - 47) - (1.25)(PaCO_2)\} - PaO_2$$

Q. What are the problems with hyperoxia?

Hyperoxia is classified as:

- Normobaric hyperoxia – caused due to breathing a high inspired concentration of oxygen at one atmosphere absolute pressure
- Hyperbaric hyperoxia – caused when breathing at high atmospheric pressure (e.g. deep sea divers). In this situation, inspired oxygen concentration may be normal (0.21) or lower but still result in an inspired PO₂ greater than that when breathing air at normal atmospheric pressure.

Hyperoxia causes cellular injury through increased production of reactive oxygen species (ROS), such as the superoxide anion, the hydroxyl radical, and hydrogen peroxide. Whenever there is imbalance between ROS and antioxidant defenses of cells, it leads to increased cell death by apoptosis and increased release of endogenous damage-associated molecular pattern molecules (DAMPs) that stimulate an inflammatory response. Hyperoxia is known to cause pulmonary oxygen toxicity, bronchopulmonary dysplasia in newborns, absorption atelectasis, aggravate bleomycin induced lung injury and retinopathy of prematurity or retrolental fibroplasia in neonates.

Q. What is the difference between actual and standard bicarbonate?

Actual bicarbonate: The actual bicarbonate is the value calculated from the blood gas sample.

Standard/corrected bicarbonate: The value obtained after correction of CO₂ levels to 40 mm Hg and at room temperature. It gives a better estimate of the metabolic problem causing acid-base imbalance.

The base deficit/excess is the amount of deviation of the standard bicarbonate from the normal.

Q. What are indications for ABG analysis?

One should understand the reason why they are obtaining any ABG. In some cases, noninvasive monitoring such as pulse oximetry, transcutaneous gas monitoring, or capnography may be sufficient. In other cases, the procedure may be reconsidered if it is not clear that the results of the test will alter the course of treatment.

Indications:

- The need to assess ventilatory status, acid-base balance, and oxygenation and the oxygen-carrying capacity of the blood;
- To assess a patient's response to therapeutic intervention like ventilator management, circulatory intervention or the progression of a disease process; and
- For surgical evaluation (pulmonary resections).

Q. Explain role of kidneys in maintaining acid-base homeostasis.

Organic and mineral acids are products of normal metabolic processes. These are immediately buffered so pH remains stable. Buffers are bicarbonate, proteins and bone.

Kidneys excrete mineral ions (associated with producing these mineral acids) and regenerate the 60–70 mEq of bicarbonate lost daily whilst buffering these acids. Kidneys do this by reabsorbing bicarbonate and producing ammonium.

Q. Is it mandatory to have arterial line for ABG sampling.

An arterial line should be placed when multiple blood gas studies (more than 4 samples of arterial blood in 24 hours) are anticipated. For less than 4 samples/24 hours, collecting sample through arterial puncture should be performed but care should be taken to change the puncture site every time as repeated puncture of a single site increases the likelihood of hematoma, scarring, or laceration of the artery.

Q. What are contraindications for arterial puncture?

- Cellulites or other infections over the puncture site of artery
- Absence of palpable arterial pulse

- Negative results of an Allen test/modified Allen test (collateral circulation test), indicating that only one artery supplies the hand/foot and suggest to select another extremity as the site for arterial puncture
- Coagulopathies or medium-to-high-dose anticoagulation therapy (e.g. heparin or Coumadin, streptokinase, and tissue plasminogen activator but not necessarily aspirin) may be a relative contraindication for arterial puncture
- History of arterial spasms following previous punctures
- Severe peripheral vascular disease
- Arterial grafts.

Q. What is the most preferred artery for puncture?

The radial artery on nondominant hand is the ideal site for an arterial puncture for the following reasons:

- It is small, but superficial and easily accessible, and stabilized
- It is easily compressible with better control of bleeding
- There is no nerve near it to worry about
- The collateral arch with ulnar artery minimizes the risk of occlusion.

Q. How will you check for adequacy of collateral circulation/What is Allen's test?

- Patient elevates hand and makes a fist for 20 seconds
- Firm pressure held against radial and ulnar arteries
- Patient opens hand and should blanch white
- Examiner releases only ulnar compression
- *Normal result:* Hand color flushes within 5 to 7 seconds
- *Abnormal result:* Delayed hand flushing indicating inadequate collateral circulation.

Alternative means for assessing collateral circulation:

- Doppler ultrasound
- Finger plethysmography.

Q. What is modified Allen's test?

This is done before puncturing dorsalis pedis artery. Elevate patient's feet. Occlude dorsalis pedis artery; then blanch the great toe by compressing the toenail for several seconds. Release pressure on the nail and observe for flushing (rapid return of color indicates adequate collateral flow). Compress the posterior tibial artery if it is being used as the puncture site (pediatric population).

Q. Describe procedure for arterial puncture.

A comfortable setting is essential to proper technique. The patient should be lying down or sitting with the arm well-supported and the clinician should also be seated if possible. Thoroughly clean the skin around the site using your institutional protocol.

A rolled towel positioned beneath the wrist helps hyperextend the site while the pulse is carefully palpated. Note that sometimes palpating too firmly can occlude the artery enough to prevent blood from flowing into the syringe, even though the artery has been punctured.

If the syringe needs to be repositioned, the tip should be withdrawn to the subcutaneous tissue to prevent damaging the artery or tendons with the needle.

Do's

- Communicate with the patient about the purpose of the procedure
- Always perform Allen test prior to drawing blood from a radial artery
- Apply pressure to the site for at least 5 minutes (normal clotting time) or more if patient has prolonged clotting time
- Keep ABG sample in an ice bag sample unless it is going to be analyzed within 10–15 minutes.

Don'ts

- Palpate too firmly, inhibiting blood flow
- Reposition a needle without first withdrawing the tip to subcutaneous tissue
- Ever leave bubbles in an ABG syringe or draw air in before deairing
- Fail to adequately heparinize a sample to prevent clotting (we get preheparinized syringes for ABG collection. Preferably use these syringes. If unavailable, take a 2 cc syringe flush it with heparine and then use for ABG collection).

List complications of arterial puncture

- Pain
- Bruising and hematoma
- Nerve damage
- Aneurysm
- Spasms
- Arteriovenous fistula
- Infection
- Vasovagal response
- Air or thromboembolism
- Anaphylaxis from local anesthetic.

Q. How would you time ABG sample?

To get the correct information about underlying pathophysiology blood sampling must be done during steady state whenever there is initiation or change in oxygen therapy or changes in ventilatory parameters with patients on mechanical ventilation. In the patients without overt pulmonary disease a steady state is reached between 3–10

minutes and in patients with chronic airways obstruction it takes about 20–30 minutes after changes have been made.

Q. How does excess heparin affect your ABG report or how will you heparinize the syringe?

Heparin must be added to the syringe as an anticoagulant. Because the pH of heparin is near 7.0, and the PO_2 and PCO_2 of the heparin solution are near room air values, excess heparin can alter all three ABG measurements. Very little heparin is actually needed in the sample to prevent clotting; 0.05 to 0.10 mL of a dilute solution (1000 units/mL) will anticoagulate 1 mL of blood without affecting its pH, PCO_2 , or PO_2 . After flushing the syringe with heparin, a sufficient amount usually remains in the dead space of the syringe and needle for anticoagulation without distortion of the ABG determination.

Q. Why proper handling of sample is necessary?

Blood is living tissue. Leukocytes and platelets continue to consume oxygen in the sample after it is drawn into a syringe and carbon dioxide continues to be produced. This can cause a significant fall in PaO_2 and rise in PCO_2 over time at room temperature, especially in the setting of leukocytosis or thrombocytosis. It is essential that the ABG sample be run within 10–15 minutes or immediately be put in a container of ice. An ABG sample can remain stable on ice for at least 1 hour. One may argue that an iced sample can remain stable for up to several hours, but at that point it is no longer representative of the patient's current status and its value as a clinical tool is severely diminished. Failure to cool the sample properly is a common source of preanalytic error.

If air bubbles are not removed immediately, oxygen can diffuse into the sample and compromise the results. Air bubbles that mix with a blood sample will result in gas equilibration between the air and the blood. Room air has a PO_2 of approximately 150 mm Hg (sea level) and a PCO_2 of essentially zero. Thus, air bubbles that mix and equilibrate with arterial blood will shift the PaO_2 toward 150 mm Hg and significantly lower the PCO_2 of the blood sample with subsequent increase in pH.

Correcting patient temperature, once commonly applied to ABG samples, especially in patients on cardiopulmonary bypass is no longer the standard as studies have failed to show much clinical relevance of temperature-corrected PO_2 values.

Q. What are commonest sources of error in ABG?

Accurate results for ABGs depend on collecting, handling, and analyzing the specimen properly. Clinically important errors may occur at any of these steps, but ABG measurements are particularly vulnerable to preanalytic errors (collection, storage and transport of sample). The most common problems include nonarterial samples, air bubbles in the sample, either inadequate or excessive

anticoagulant in the sample, and delayed analysis of an uncooled sample.

Q. How will you choose the site?

Arterial blood samples are normally obtained from adults at the radial, brachial, femoral, or dorsalis pedis arteries. Because radial artery puncture is relatively safe and the site easily accessible as well as convenient for checking collateral circulation, this site is preferable. Before doing arterial puncture one should test for collateral circulation. If collateral circulation is absent, the radial artery should never be used.

The brachial artery is the second choice, as it is relatively large and easy to palpate, and has good collateral circulation; however, it lies deeper and its proximity to the basilic vein and median nerve makes it easy to hit them by mistake. In addition, the lack of underlying ligaments or bone support increases the risk of hematoma following the procedure.

The femoral artery is the third choice because it is relatively easy to palpate and it is sometimes the only site where sampling will be possible; however, it lies close to the femoral vein, poses increased risk of infection, and requires prolonged monitoring after puncture. It should be selected as a last resort and only within a hospital setting.

Dorsalis pedis artery is also a useful site for ABG punctures when a radial artery sample is not obtainable.

Q. How do calculate oxygen content of blood?

Oxygen content can be measured directly or calculated by the oxygen content equation:

$$CaO_2 = (Hb \times 1.34 \times SaO_2) + (0.003 \times PaO_2)$$

Q. What are the different methods of analyzing acid-base disorders?

There are two methods of evaluation of acid-base disorders:

1. *Classic or traditional approach:* It is bicarbonate based approach essentially revolves around the Henderson-Hasselbalch formula:

$$pH = pK + \log \frac{[HCO_3]}{PCO_2}$$

Respiratory disorders are due to a change in PCO_2 whereas metabolic disorders are due to alterations in bicarbonate. Based on this approach acid-base disorders are classified into 6 primary disorders:

- Metabolic acidosis (high anion gap/normal anion gap)
 - Metabolic alkalosis
 - Acute and chronic respiratory acidosis
 - Acute and chronic respiratory alkalosis.
2. *Stewart approach:* This approach is independent of the bicarbonate.

Its variables are pCO_2 , strong ion difference (SID) and total weak acids (ATOT)

Respiratory disorders are due to alterations in $p\text{CO}_2$ similar to the traditional approach.

Metabolic disorders are due to primary alterations in strong ion difference (SID) and strong ion gap (SIG) or ATOT and not bicarbonate (**Table 1**):

- The cations are Na^+ , K^+ , Ca^{++} and Mg^{++}
- The anions are Cl^- , Lactates and other strong ions
- $\text{SID apparent} = [\text{Na}^+ \text{K}^+ \text{Ca}^{++} \text{Mg}^{++}] - [\text{Cl}^-]$
- Normal strong ion difference is about 40 mEq/L
- $\text{SID effective} = [\text{HCO}_3^-] + [\text{A}^-]$
- Strong ion gap (SIG) = $\text{SIDa} - \text{SIDe}$ normal strong ion gap is zero
- ATOT represents all nonbicarbonate buffer pairs primarily albumin, inorganic phosphate, hemoglobin
- $\text{ATOT} = [\text{HA}] + [\text{A}^-]$.

TABLE 1: Stewart approach.

Parameter		Acidosis	Alkalosis
Respiratory		$\uparrow \text{PCO}_2$	$\downarrow \text{PCO}_2$
Nonrespiratory (metabolic)			
SID	Abnormal SID	$\downarrow \text{SID}$	$\uparrow \text{SID}$
	Anion excess	$\downarrow \text{SID} \uparrow$ Lactates	
ATOT (nonvolatile weak acids)	Albumin	$\uparrow$	$\downarrow$
	Inorganic phosphate	$\uparrow$	$\downarrow$

(SID: strong ion difference)

Q. What are normal values in ABG?

Table 2 shows the normal values in ABG.

TABLE 2: Normal values in ABG.

Sample	PaO_2 (mm Hg)	PaCO_2 (mm Hg)	pH	HCO_3^- mmol/L	Base excess	SaO_2
Arterial	80–100	35–45	7.36– 7.44	22–26	+/- 2	94– 100
Venous	37–42	42–50	7.34– 7.42			
Normal range						
Anion gap			12–16 mmol/L			
Osmolar gap			10 mmol/L			
$\text{PaO}_2/\text{FiO}_2$ ratio			>300			

Q. What is compensation? State the rules of compensation and expected compensation in various disorders.

Maintenance of acid-base balance is normally accomplished by buffer systems which react to changes in the hydrogen ion concentration of body fluids. When there is an

imbalance in one parameter, the other tries to compensate for it by causing the opposite imbalance. The goal of this compensation process is to restore the 20:1 (bicarbonate to carbonic acid) ratio, and return the pH back to the acceptable range of 7.35–7.45.

Rules of Compensation

- The compensatory response depends upon the proper functioning of organ system involved in the response (lungs or kidneys) and on the severity of acid-base disturbance.
- Acute compensation in case of kidneys occurs within 6–24 hours and chronic within 1–4 days. Respiratory compensation occurs faster than metabolic compensation.
- In clinical practice, it is uncommon to see complete compensation. The maximum compensatory response in most of the cases is associated with only 50–75% return of pH to normal.

Expected compensation for simple acid-base disorders

- **Metabolic acidosis:** Expected PaCO_2 decreases by 1–1.3 mm Hg for every 1 mEq/L decrease in HCO_3^- or expected PaCO_2 = last two digits of pH.
- **Metabolic alkalosis:** Expected PaCO_2 increases by 0.5–0.7 mm Hg for every 1 mEq/L increase in HCO_3^- or expected PaCO_2 = last two digit of pH.
- **Respiratory acidosis:** In acute acidosis, HCO_3^- increases by 1 mEq/L for every 10 mm Hg increase in PaCO_2 . In chronic acidosis, the HCO_3^- increases by 3.5 mEq/L for each 10 mm Hg increase in PaCO_2 .
- **Respiratory alkalosis:** In acute alkalosis, HCO_3^- decreases by 2 mEq/L for every 10 mm Hg decrease in PaCO_2 . In chronic alkalosis, HCO_3^- decreases by 5 mEq/L for every 10 mm Hg decrease in PaCO_2 .

Q. How do you determine if compensation is present, and to what extent?

When compensation is present, we will see two imbalances. The question then becomes, which is the primary problem, and which imbalance is due to compensation. The clue is the pH. If the pH is leaning toward acidosis or alkalosis, then the parameter with the matching imbalance is the primary problem, and the other is due to compensation. Also knowing patients proper history will help us identify the primary pathology.

Single acid-base disorders do not lead to normal blood pH. Although pH can end up in the normal range (7.35–7.45) with a single mild acid-base disorder, a truly normal pH with distinctly abnormal HCO_3^- and PaCO_2 invariably suggests two or more primary disorders.

For example 7.43 (within acceptable range, but not perfectly normal 7.4) $\text{PCO}_2 = 20$, $\text{HCO}_3^- = 15$.

Interpretation: The pH is alkaline, so the primary problem is alkalosis. Respiratory alkalosis with nearly complete compensation by the kidneys.

7.36 (within acceptable range, but not perfectly normal 7.4) $\text{PCO}_2 = 10$, $\text{HCO}_3 = 5$.

Interpretation: The pH is acidotic, so the primary problem is acidosis. Metabolic acidosis with compensation by the lungs.

Q. What are types of respiratory failure and causes of respiratory failure?

Type I respiratory failures are also known as hypoxemic failures and type II as hypercapnic failures (**Table 3**).

TABLE 3: Types of respiratory failure.

Type I	Type II
Atelectasis	Central nervous system depression (drugs/sleep/head injury)
Pulmonary edema (cardiogenic/noncardiogenic)	High spinal cord lesion
Pneumonia	Phrenic nerve lesions
Pleural effusion	Neuromuscular disorders
Hemopneumothorax	Severe kyphoscoliosis
	Chronic obstructive pulmonary disease
	Type I causes in advanced states

Q. How is metabolic acidosis produced?

Metabolic acidosis is produced when:

- Acid production is in excess of kidney ability to excrete acid and regenerate bicarbonate
- Increased loss of bicarbonate from extracellular fluid (e.g. GIT loss or renal)
- Renal pathology causing reduction in ability to excrete acid and regenerate bicarbonate.

Q. What do you understand by anion gap?

Anion gap is difference between measured cations and measured anion. There is no difference in actual cations and actual anions. Body fluids including blood may contain a variable number of ions, but the total number of anions (negative ions) and cations (positive ions) are roughly the same. The ions that are usually measured in blood are cations such as sodium and potassium and anions including chloride and bicarbonate. There are unmeasured ions in both groups (cations and anions), which also contribute to the ionic constitution of blood. The measured cations are usually greater than the measured anions by about 8–12 mmol/L. This is because the unmeasured anions constitute a significant proportion of the total number of anions in blood. Proteins make this up predominantly, but also included are sulfates, phosphates, lactate and ketones.

Anion gap = $(\text{Na} + \text{K}) - (\text{Cl} + \text{HCO}_3)$ Normal AG is 14–16 if we include potassium.

Also, anion gap = $(\text{Na}) - (\text{Cl} + \text{HCO}_3)$ Normal AG is 8–12 if we do not include potassium.

Causes of metabolic acidosis:

High anion gap: KULT or MUDPILES

- Ketoacidosis diabetic:
 - Starvation
 - Alcoholic (ethanol)

- Lactic acidosis

- Uremia

- Toxins

Methanol:

- Ethylene glycol
- Propylene glycol
- Salicylates
- Paraldehyde

Normal anion gap acidosis

- Hypokalemic:
 - Gastrointestinal losses of HCO_3 :
 - Ureteral diversion
 - Diarrhea
 - Ileostomy
 - Renal losses of HCO_3 :
 - Proximal renal tubular acidosis (RTA)
 - Carbonic anhydrase inhibitors
- Normokalemic/hyperkalemic:
 - Renal tubular disease:
 - Acute tubular necrosis
 - Chronic tubulointerstitial disease
 - Distal RTA (type I and IV)
 - Hypoaldosteronism
 - Pharmacological:
 - Ammonium chloride
 - Hyperalimentation
 - Dilutional acidosis

Decreased anion gap acidosis:

- Hypoalbuminemia
- Paraproteinemia (multiple myeloma)
- Spurious hyperchloremia
- Bromide intoxication
- Spurious hyponatremia
- Hypermagnesemia.

Q. What is corrected anion gap?

Critically ill patients generally are hypoalbuminemic and hypophosphatemic. Albumin is a major contributor for anion gap. In the presence of low serum albumin, anion gap is reduced by approx 2.5 mEq/L for every 1 g/dL fall in albumin. Alternatively, a corrected value for a normal

anion gap (assuming K is included in calculation of anion gap) can be obtained from:

Corrected normal anion gap = $0.2 (\text{albumin}) \times 1.5 (\text{phosphate})$ where albumin is in g/dL and phosphate in mmol/L.

Also, corrected anion gap = Observed anion gap + $2.5 \times (\text{Normal albumin} - \text{Observed albumin})$

Normal albumin is taken as 4.4 g/dL.

Q. How do you treat metabolic acidosis?

One should always be cautious when interpreting any result. The aim should not be to correct a number but the underlying abnormality that is represented by abnormal number. Metabolic acidosis points towards a specific underlying pathology which needs to be addressed. For example, raised lactic acidosis mostly suggests a hypoperfusion state. So, treatment should be directed towards improving perfusion by optimizing fluid status and using inotropes rather than giving bicarbonate.

Indications for bicarbonate therapy in acidosis:

- pH < 7.2 and bicarb < 5 mEq/L
- Patients with bicarbonate loss (normal anion gap acidosis)
- Not indicated in raised AG acidosis, treat underlying pathology.

Complications of bicarbonate therapy:

- Volume overload
- Hyponatremia
- Hyperosmolarity
- Hypokalemia
- Intracellular acidosis
- Overshoot alkalosis
- Shift of oxyhemoglobin dissociation curve to left.

Q. What is urine anion gap?

The urine anion gap (UAG) helps in defining the cause in case of normal anion gap acidosis.

It is defined as $\text{Na}^+ \text{K}^+ \text{Cl}^-$ in urine.

The UAG is normally negative as a result of excretion of ammonium into urine. If UAG is positive it reflects an impairment of ammonium excretion as seen in patients with renal tubular acidosis (RTA). Urinary pH then further helps in knowing type of RTA. If pH is >6 a distal RTA is present and if pH is low and remains low in spite of bicarbonate infusion proximal RTA is the underlying pathology.

Q. What are the causes of metabolic alkalosis and how do you treat it?

Causes of metabolic alkalosis:

- Loss of H^+ ions (e.g. vomiting, diuretics)
- Increased reabsorption of bicarbonate:
 - Low intravascular volume

- Hypokalemia
- High pCO_2
- Increased mineralocorticoids (aldosterone).

- Administration of alkali (in setting of renal impairment), e.g. Ringer's lactate where lactate gets metabolized to bicarbonates in liver adding to alkali pool.

Treatment

- Correct dehydration (intravascular volume) with chloride rich fluid and potassium
- If the patient has CCF (or primary excess mineralocorticoids) use aldosterone antagonist (spironolactone)
- Use of carbonic anhydrase inhibitors like acetazolamide to enhance loss of bicarbonates from kidneys.

Q. What are causes of respiratory acidosis?

Any condition that results in hypoventilation can cause respiratory acidosis. These conditions include:

- Airway/pulmonary parenchymal disease:
 - Upper airway obstruction
 - Lower airway obstruction
 - Pulmonary alveolar process:
 - Cardiogenic pulmonary edema
 - Pneumonia
 - Acute respiratory distress syndrome (ARDS)
 - Pulmonary perfusion defect—PE—air/fat/tumor
- Normal airway/pulmonary parenchymal:
 - Central nervous system depression
 - Central nervous system depression related to head injury
 - Central nervous system depression related to medications such as narcotics, sedatives, or anesthesia
- Neuromuscular disease and impairment: Impaired respiratory muscle function related to spinal cord injury, neuromuscular diseases, or neuromuscular blocking drugs
- Ventilatory restriction: Due to pain, chest wall injury/deformity, or abdominal distension.

Q. What are causes of respiratory alkalosis?

Any condition that causes hyperventilation can result in respiratory alkalosis. These conditions include:

- CNS stimulation: Fever, pain, thyrotoxicosis, cerebrovascular accidents
- Hypoxemia: Pneumonia, pulmonary edema, severe anemia
- Drugs/hormones: Medroxyprogesterone, catecholamines, salicylates
- Miscellaneous: Sepsis, pregnancy
- Psychological responses, such as anxiety or fear.

Q. What happens to serum sodium level in hyperglycemia and how do you calculate correct sodium in presence of hyperglycemia?

Since, normal blood glucose is relatively low its contribution of osmotic pressure is relatively insignificant. However in presence of severe hyperglycemia as may be seen in uncontrolled diabetics, blood glucose exerts significant osmotic pressure resulting in raised serum osmolality that leads to shift of water from intracellular to extracellular compartment. This will dilute electrolytes in blood and as a result serum sodium concentration appears to be low. This is called as fictitious hyponatremia. In presence of significant hyperglycemia one can calculate corrected serum sodium as follows:

Corrected sodium = Measured Na + $[(BS \text{ in mg\%} - 100)/100] \times 1.4$

Q. What is osmolar gap and what are the causes?

The plasma osmolality can be calculated by the formula Plasma osmolality = $2 \times \text{Sodium (mEq/L)} + \text{Glucose (mg/dL)}/18 + \text{Urea (mg/dL)}/2.8$. If the calculated osmolality differ from the measured osmolality by 15 mOsm/kg H₂O, this is called as osmolar gap and further investigations need to be done. Causes are:

- Ethanol
- Isopropyl alcohol
- Methanol
- Glycine
- Mannitol
- Ethylene glycol
- Glycerol
- Chronic renal failure.

Q. A 55-year-old man presents with shortness of breath. He is hypertensive on chlorothiazide and aspirin. He is chronic smoker with 60 pack year's history.

On room air his ABG shows pH 7.53, PaO₂ 62 mm Hg, PaCO₂ 37 mm Hg, HCO₃⁻ 30 mEq/L Hb 14 g% and SaO₂ 87%. How would you characterize his state of oxygenation, ventilation, and acid-base balance?

Oxygenation: The PaO₂ and SaO₂ are both reduced on room air. Since, P(A-a)O₂ is elevated (approximately 43 mm Hg), the low PaO₂ can be attributed to V-Q mismatch, i.e. a pulmonary problem. SaO₂ is reduced, in part from the low PaO₂ and from elevated carboxyhemoglobin as he is a smoker. The arterial oxygen content is adequate (CaO₂ 16.5 mL O₂/dL).

Ventilation: Adequate for the patient's level of CO₂ production; the patient is neither hyper- nor hypoventilating.

Acid-base: Elevated pH and HCO₃⁻ suggest a state of uncompensated metabolic alkalosis, most likely related to

the patient's diuretic; his serum K⁺ should be checked for hypokalemia.

Q. A 46-year-old man has been in the hospital two days with pneumonia. He was recovering but has just become diaphoretic, dyspneic, and hypotensive. He is breathing oxygen through a nasal cannula at 3 L/min.

pH 7.40 PaCO₂ 20 mm Hg PaO₂ 80 mm Hg SaO₂ 95% Hb 13.3 gm% HCO₃⁻ 12 mEq/L. How would you characterize his state of oxygenation, ventilation, and acid-base balance?

Oxygenation: The PaO₂ is lower than expected for someone of this age and receiving supplemental oxygen, and points to significant V-Q imbalance. The oxygen content is adequate. (CaO₂ 17.2 mL O₂/dL).

Ventilation: PaCO₂ hyperventilation is half normal and indicates marked hyperventilation.

Acid-base: Normal pH with very low bicarbonate and PaCO₂ indicates combined respiratory alkalosis and metabolic acidosis.

Q. Which is primary pathology?

Patient has deteriorated acutely. Only compensation that works faster is respiratory. Metabolic full compensation takes 2-3 days. Since compensation is full, compensating process is respiratory and primary pathology is metabolic. Patient is hypotensive hence sepsis should be strongly considered, especially in someone with a documented infection.

Q. What are mixed acid-base disorders and how do you diagnose them?

The simple, or primary, acid-base disorders (respiratory and metabolic acidosis and alkalosis) evoke a compensatory response that produces a secondary acid-base disturbance and reversion of the blood pH towards (rarely to) normal, e.g. a simple metabolic acidosis will result in a secondary respiratory alkalosis, both of which will ordinarily be reflected in the patients' acid-base-related analysis in blood. When two primary acid-base disturbances arise simultaneously in the same patient, the complex is called a mixed acid-base disorder. If three primary disturbances occur together, the patient is described as having "triple acid-base disorder."

For example, the development of a primary metabolic alkalosis in a patient with chronic obstructive airway disease who is being treated with diuretics should alert the clinician to the possibility of potassium depletion. To diagnose them always follow following rules:

- Do not interpret any blood gas data for acid-base diagnosis without also examining the corresponding serum electrolytes

- Single acid-base disorders do not lead to normal blood pH. Although, pH can end-up in the normal range (7.35 – 7.45)
- Simplified rules predict the pH and HCO_3^- —for a given change in PaCO_2 . If the pH or HCO_3^- is higher or lower than expected for the change in PaCO_2 , the patient probably has a metabolic acid-base disorder as well.
- In maximally-compensated metabolic acidosis (which takes about 12–24 hours), the following formula applies: Expected $\text{PaCO}_2 = \text{last two digits of pH} \pm 2$.

In contrast, the compensation for metabolic alkalosis (by increasing PaCO_2) is highly variable, and in some cases there may be no or minimal compensation.

Q. A 50-year-old woman who presented semi-comatose at the emergency department. She had been receiving insulin therapy for diabetes mellitus, and over the previous four months had been taking digoxin and chlorothiazide tablets for congestive cardiac failure.

Her ABG shows pH 7.41 PaCO_2 32 mm Hg PaO_2 88 mm Hg SaO_2 95% Hb 13.3 g% HCO_3^- 19 mEq/L, K 2.7 mEq/L, Na 132 mEq/L, Cl 79 mEq/L and blood sugar 88 mg%. Urine ketones are positive:

- Oxygenation: OK
- Ventilation: Slightly hyperventilating
- Acid-base.

The diagnostic feature of this patient's biochemistry values is the combination of mild metabolic acidosis HCO_3^- of 19 mmol/L and a very high anion gap (36 mmol/L). If we assume that the normal anion gap for this patient was at the upper limit of the reference interval 16 mmol/L, then the increase in her gap is 20 mmol/L. If this increase was due to diabetic-induced ketosis—the obvious explanation—then the approximate decrease in her plasma HCO_3^- would be of the same magnitude. Thus, her original concentration of plasma bicarbonate would have been on the order of 39 mmol/L ($19 + 20$ mmol/L), i.e. metabolic alkalosis. Inspection of her clinical history and of the concentration of potassium in her plasma indicates that she presumably had a metabolic alkalosis, from thiazide therapy and hypokalemia, before the diabetes induced ketoacidosis.

Diagnosis: Mixed metabolic alkalosis and metabolic acidosis.

Q. A 24-year-old with insulin dependent diabetes presents to emergency department with cough, fever and breathlessness.

Her ABG: pH 7.33, PCO_2 25, HCO_3^- 12 PaO_2 79 with BS 780 mg%

Diagnosis: Metabolic acidosis with respiratory alkalosis
metabolic acidosis expected $\text{PaCO}_2 = \text{last two digits of pH}$.

You expect her PaCO_2 around 30–33 mm Hg. Here it is lower suggesting combined metabolic acidosis with respiratory alkalosis.

Her PaO_2 is low and she gives history of cough and fever. Probably, she has pneumonia and needs to get investigated further along with control of diabetes.

Q. What do you understand by validation of ABG and why do you need it?

This was an important step in old times when there were no printers attached to ABG machine and person doing ABG would write down the values from machine screen. Errors (transcription error) could occur while writing down these values and could significantly affect the ABG interpretation. However, in modern days with ABG machine fitted with printer this is no more a problem and one need not validate the report. However one has to remember that in the absence of a printed report (e.g. verbally conveyed) you may have to confirm validity.

This can be done using following formula:

$$\text{H} \times \text{HCO}_3^- / \text{PaCO}_2 = 24 (+/- 2 \text{ nmol/L})$$

To calculate H^+ in mEq/L from pH, use the following table.

pH	6.80	6.90	7.00	7.10	7.20	7.30	7.40	7.50	7.60	7.70	7.80
(-) from			100	90	80				85	90	95
(H^+)	160	130	100	80	60	50	40	30	25	20	15

Q. Enumerate the steps for analyzing the ABG.

Step 1: Look at the pH.

Is the patient acidemic ($\text{H} < 7.35$) or alkalemic ($\text{pH} > 7.45$)

Step 2: Is it a primary metabolic or respiratory disturbance?

Acidemia: With $\text{HCO}_3^- < 20$ mmol/L = metabolic. With $\text{PCO}_2 > 45$ mm Hg = respiratory

Alkalemia: With $\text{HCO}_3^- > 28$ mmol/L = metabolic. With $\text{PCO}_2 < 35$ mm Hg = respiratory

Go to step 3 and 4 if respiratory and step 5 to 7 if metabolic.

Step 3: If there is a primary respiratory disturbance, it is acute? Expect $\Delta \text{pH} = 0.08 \times \Delta \text{PCO}_2 / 10$

[For respiratory acidosis ($\text{PCO}_2 - 40$) = ΔPCO_2]

[For respiratory alkalosis ($40 - \text{PCO}_2$) = ΔPCO_2]

If not acute go to step 4, if yes—also consider steps 5–7.

Step 4: For a respiratory disorder is renal compensation OK?
Respiratory alkalosis:

- <24 hours: $\Delta[\text{HCO}_3^-] = 1/10 \times \Delta \text{PCO}_2$

- >24 hours: $\Delta[\text{HCO}_3^-] = 4/10 \times \Delta \text{PCO}_2$

Respiratory acidosis:

- 1–2 hours: $\Delta[\text{HCO}_3^-] = 2/10 \times \Delta \text{PCO}_2$

- >2 days: $\Delta[\text{HCO}_3^-] = 5/10 \times \Delta \text{PCO}_2$

Step 5: If the disturbance is metabolic is the respiratory. Compensation appropriate?

For metabolic acidosis:

$$\text{Expect } \text{PCO}_2 = [1.5 \times (\text{HCO}_3)] + 8 \pm 2$$

For metabolic alkalosis:

$$\text{Expect } \text{PCO}_2 = [(0.7 \times (\text{HCO}_3)] + 21 \pm 1.5$$

If not actual $\text{PCO}_2 > \text{expected}$: Hidden respiratory acidosis, actual $\text{PCO}_2 < \text{expected}$: Hidden respiratory alkalosis.

Step 6: If there is metabolic acidosis, is there an anion gap? $\text{Na} - (\text{Cl} + \text{HCO}_3) = \text{Anion gap}$ usually < 12 . If gap is more than 12, patient has an anion gap acidosis.

The usual causes are remembered by MUDPIES:

- Methanol
- Uremia
- Diabetic ketoacidosis
- Paraldehyde
- Infection (lactic acid)
- Ethylene glycol
- Salicylate.

Step 7: Does the anion gap explain the change in bicarbonate? $[(\text{HCO}_3) + (\text{gap} - 12)] = 24$

- If parameter is greater than 24; Consider additional hidden metabolic alkalosis.
- If parameter is less than 24; Consider a hidden nonanion gap metabolic acidosis.
- This is valid only in patients who do not have a chronic respiratory acid-base disorder.

Q. A 45-year-old male is admitted to the ICU with pancreatitis. His ABG is, pH = 7.33, $\text{PaCO}_2 = 30$ mm Hg, $\text{HCO}_3 = 15$, lactates = 6, albumin = 2 g/dL, Na = 136, K = 4.5,

Cl = 108, Ca = 3.3, Mg = 1.6 (mEq/L), $\text{PO}_4 = 2$ mmol/L. Interpret it using traditional and the Stewart approach.

- Using the classic approach:
 - pH = 7.33 is suggestive of acidosis, HCO_3 of 15 is suggest metabolic acidosis with a compensatory fall in CO_2 because of hyperventilation
 - Anion gap = $[\text{Na}] - [\text{Cl} + \text{HCO}_3] = 136 - 123 = 13$
 - Corrected anion gap for albumin = observed AG + 2.5 (Normal albumin - observed albumin) = $13 + 2.5 (4.4 - 2) = 13 + 6 = 19$
 - Hence, this is a high anion gap metabolic acidosis due to elevated lactates.
- Using the Stewart approach:
 - PH = 7.33 indicates acidosis
 - $\text{SID} = [\text{Na} + \text{K} + \text{Ca} + \text{Mg}] - [\text{Cl}]$
 $= [136 + 4.5 + 3.3 + 1.6] - [108] = 145.4 - 108 = 37.4$
 - Since, SID is less than 40 this is suggestive of metabolic acidosis with anion excess-lactates.

SUGGESTED READING

1. Barthwal MS. Analysis of arterial blood gases: a comprehensive approach. J Assoc Physicians India. 2004;52:573-7.
2. Corey HE. Bench-to-bedside review: fundamental principles of acid-base physiology. Crit Care. 2005;9:184-92
3. Gattinoni L, Carlesso E, Cadringer P, Caironi P. Strong ion difference in urine: new perspectives in acid-base assessment. Crit Care. 2006;10:137.
4. Kellum JA. Disorders of acid-base balance. Crit Care Med; 2007.
5. Rastegar A. Clinical utility of Stewart's method in diagnosis and management of acid-base disorders. Clin J Am Soc Nephrol. 2009;4:1267-74.
6. Sood P, Paul G, Puri S. Interpretation of arterial blood gas. Indian J Crit Care Med. 2010;14:57-64.

Mechanical Ventilation

Atul Prabhakar Kulkarni, Sohan Lal Solanki, Jigeeshu Vasistha Divatia

Q. How are ventilators classified?

Ans. There are numerous ways to classify ventilators.

Single Circuit versus Double Circuit Ventilators

Ventilators that mix and deliver compressed gas directly to the patient are single circuit ventilators. They expel the expired gases to the atmosphere during expiration. Ventilators used in critical care are of this type. A double circuit ventilator is analogous to a reservoir bag powered by a single circuit ventilator. These ventilators deliver a driving gas, usually compressed oxygen or air, to a bellows assembly. The driving gas pressurizes the rigid space outside the bellows, causing the bellows to squeeze its contents of patient gas into the breathing circuit and the patient's lungs. For example, North American Drager and Ohmeda anesthesia ventilators.

Classification According to their Power

- *Low-powered ventilators:* These generate only modest gas pressures required to deliver reasonable tidal volumes to the lungs (in a normal or near-normal lung with respect to resistances and compliances). Their use is limited as in a damaged lung the tidal volume delivered will be insufficient. Thus, when these ventilators are used, monitoring of the ventilation becomes mandatory.
- *High-powered ventilators:* A ventilator needs to be powerful to maintain its performance in deteriorating lung conditions like changes in lung-thoracic compliance and airway resistances. Up to 80 cm of H₂O is sufficient without alteration in gas flow (flow generators). These require a safety valve (pressure relief valve) which can be preset or adjustable. They may also require a flow restrictor to avoid build-up of high pressure in the lungs.

Classification According to Cycling

A ventilator is said to cycle in two phases—inspiratory and expiratory:

1. *Inspiratory cycling:*

- *Volume cycling:* These ventilators recognize the point at which the predetermined volume of the gas has left the ventilator and switches its internal mechanism to allow exhalation to occur.
- *Time cycling:* These use mechanical, pneumatic or electronic timers to operate the cycling mechanism, which work independently of the delivered tidal volume. It may allow the tidal volume to be delivered early in the inspiratory cycle, followed by a pause to allow better distribution of the gases prior to the start of the expiratory phase.
- *Pressure cycling:* Here the inspiratory phase is terminated when the predetermined pressure is reached.
- *Flow cycling:* Flow pattern changes are used to cycle the ventilator. Now rarely used.

2. *Expiratory cycling:*

- *Volume cycling:* Here the expiratory phase is terminated when the bellows have filled to the desired tidal volume required for the next inspiration.
- *Time cycling:* It is the most popular and the most versatile method of controlling the expiratory phase. It uses electronic or pneumatic timers.
- *Pressure cycling:* It identifies a selected airway pressure at end of exhalation that would trigger the next inspiration.
- *Flow cycling:* It senses the end of exhalation when the desired flow rate is reached and then switches to inspiration.

Classification According to Application

- *Mechanical thumbs:* It is a simple T-piece system, the open end of which is occluded with the anesthetist's thumb thus inflating the patient's lungs. On release of the thumb pressure, expiration occurs passively. Thus, rhythmically intermittent positive pressure can be achieved. In ventilators like Sheffield and Amsterdam,

the thumb is replaced by an electrical solenoid valve, which is cycled by an electronic circuit.

- **Minute volume dividers:** These derive their power from the pressure of gases from the outlet of anesthetic machine. The whole of the driving gas is delivered to the patient and there is no rebreathing. Principle—A reservoir R which is continually pressurized by a spring or a weight or its own elastic recoil is continuously being filled by the fresh gas flow. Two valves, V1 and V2 are linked together and operated by a bistable mechanism. When V1 is open and V2 closes, the reservoir discharges gas to the patient (inspiration). When V1 is closed and V2 opens, expiration occurs. For example, East-Freeman automatic vent, Manley MP2, MN2 and MP3.
- **Bag squeezers:** This is a bag in bottle type of ventilator (double circuit type of ventilators). The bag or bellows can be squeezed mechanically (gears or lever or motors) or pneumatically. For example, Cape ventilator, Manley Servovent, Oxford, Penlon.
- **Intermittent blowers:** These are driven by a continuous flow of gases and air pressure of 45–60 psi is required. For example, Bird Mark 7, Penlon-AP.

Q. Discuss different types of bellows in anesthesia ventilators with their advantages and disadvantages.

Ans. Automatic ventilators include bellows that expand (fill with gas) during expiration and are compressed (to deliver gas) during inspiration in response to cycling ventilator drive pressure. Some units have ascending (rising or upright) bellows, which are attached to the base of the bellows housing and expand upward. Other units have descending (hanging) bellows, which are suspended from the top of the bellows housing and expand downward.

For an ascending bellows to expand, the expiratory pressure provided by the patient's passive lung and chest wall recoil must be sufficient to overcome the weight of the bellows. Conversely, the weight of a descending bellows assists expansion, allowing the bellows to expand—and actually draw in gas—even when no expiratory pressure is produced by the patient. Thus, when a leak or disconnection is present in the breathing circuit, a descending bellows will still expand (drawing air from the atmosphere through the leak and into the bellows) and be compressed as the ventilator cycles.

The disadvantages of the descending bellows are unrecognized disconnection (due to their design, they may fill even when disconnected from the patient), and also collection of exhaled humidity in bellows; risking infection and lessening delivered tidal volume. The modern type is ascending. Only one current machine, the Dräger Julian, uses a hanging bellows, but incorporates capnography and sensors to detect failure of the bellows to fill, both of which may lessen unrecognized disconnects.

To remember the classification: “Ascend” and “descend” have “e” in them—so look at them during expiration. Ascending bellows (“standing”) ascend during expiration (modern type—preferred by many) and descending bellows (“hanging”) descend during expiration. Ventilator relief valve gives 2–3 cmH₂O pressure positive end-expiratory pressure (PEEP). This is true for almost all mechanical ventilators—exceptions are the new Dräger Divan ventilator, which has a horizontal piston and the Julian hanging bellows. The ventilator relief valve (spill valve) only allows scavenging during expiratory phase.

The hanging design was chosen for the Julian for compactness and ease of sterilization of the entire breathing circuit. The Julian hanging bellows housing, unlike older designs, lacks an internal weight, and senses when the bellows do not return to the full “down” position. These factors, plus integration of disconnect alarms based on chemical (capnograph), and mechanical (pressure, volume, and flow sensors) detection, make piston or hanging bellows designs safe. The placement of the hanging bellows below the writing surface makes visual detection of disconnects difficult; also it is less easy to determine if the patient is breathing spontaneously in addition to the rate set on the mechanical ventilator. The user must rely more on the pressure and capnography waveforms as opposed to the bellows. Water may gather in the bellows (lessening tidal volume and creating an infection risk), but this tendency should be opposed by the heated absorber head.

Q. How does a positive pressure ventilator work?

Ans. A positive pressure ventilator is essentially just an air compressor and two valves. It pushes air in during inspiration and expiration occurs passively. That means during inspiration the inspiratory valve is open and the expiratory valve is closed. While the reverse happens during expiration; the expiratory valve opens, at the level of applied PEEP with a PEEP valve and the inspiratory valve is closed. If you have applied an inspiratory plateau period, then both valves are closed and the lung is held distended at the pressure which is the true plateau pressure (Pplat). This reflects the state of alveoli.

Q. What is a mode of ventilation?

Ans. A mode is a particular pattern of spontaneous and mandatory breaths delivered by the ventilator. Common modes include pressure support, volume control, and pressure control ventilation. There are multiple different “modes” of ventilation, all with different names and initials.

Q. What are the types of breaths?

Ans.

- **Controlled breath:** Breath initiated and delivered by the ventilator.

- *Assisted breath:* Breath initiated by the patient and assisted to the set target (volume/pressure) parameter by the ventilator.
- *Spontaneous breath:* Fully spontaneous breath, no ventilator assistance.

Q. What are the phases of respiration during mechanical ventilation?

Ans.

Phases of ventilation: A breath during mechanical ventilation can be divided into four phases. All modes can be described in terms of these phases. Once we understand these phases, it is easy to understand even newer and more complicated modes:

1. *Expiratory phase:* During this phase, the inspiratory valve is closed, and expiratory valve is open and lungs empty passively. This can be further divided into time when there is no flow (expiratory pause) and when expiratory flow takes place. PEEP is applied throughout the ventilatory cycle, including during expiration. Continuous positive airway pressure (CPAP) is nothing but PEEP during spontaneous breathing.
2. *Changeover from expiration to inspiration:* This tells the ventilator that expiration is over and inspiration should begin. The process is called triggering. Inspiration may be triggered or initiated by the patient during an assisted or spontaneous breath, or by the ventilator (controlled breath). During assisted or spontaneous breathing “triggering” allows the ventilator to sense the patient’s inspiratory effort. The two common ways by which ventilators do this are pressure and flow triggering. Pressure triggering: Patient needs to generate a small negative inspiratory pressure (generally negative 1–3 cmH₂O), which is sensed by the ventilator, and it changes over to inspiration and delivers the next breath. Flow triggering: Here a constant flow is provided in the patient circuit during expiration, flow rate being measured upstream and downstream of the patient. When the patient begins a breath the downstream flow decreases relative to the upstream flow. The ventilator senses this difference in flow and an inspiration is triggered. Flow triggering is often set in the range of 1.5–3 L/min. Flow triggering may require less work for the patient when compared to pressure triggering. During controlled ventilation, the inspiration is time-triggered, i.e., it begins after a set time. Other ways of triggering a breath are volume, impedance or motion triggering. Trigger sensitivity denotes how easily a breath may be triggered by the patient. Too low trigger sensitivity may lead to autocycling or autotriggering, which is breath delivery without effort from the patient. If the trigger is too high, the patient may waste effort without getting

a breath, leading to respiratory muscle fatigue. Wrong trigger setting can lead to patient-ventilator asynchrony.

3. *Inspiratory phase:* This phase begins with the initiation of the breath, and ends when the ventilator cycles into expiration. The peak inspiratory flow rate (PIFR) and inspiratory time settings determine time available for expiration.

Limit: During the inspiratory phase, the inspiratory valve opens, and pressure, volume, and flow all rise above their end-expiratory phase. If one (or more) variable rises no higher than a preset value, it is called a limit variable. Limit variable is the one which limits the way gas keeps flowing in the lungs during inspiration. However, the limit variable does not terminate the inspiration (that is known as cycling—**Fig. 1**); it allows inspiration to continue till the cycling criterion is reached, but at the set limit. This phase may also contain the inspiratory pause, during which the inspiratory valve and expiratory valve are both closed. During the inspiratory pause, there is no gas flow in or out of lungs and the pressure during this phase denotes the alveolar distending pressure. This pressure is also called as plateau pressure.

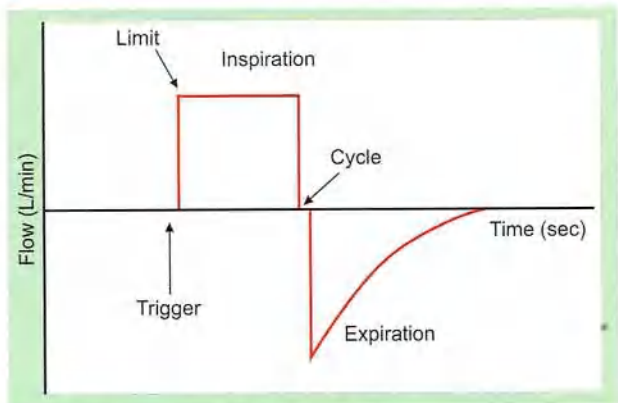


Fig. 1: Phases of respiration during mechanical ventilation.

4. *Changeover from inspiration to expiration:* This occurs when the ventilator recognizes that the inspiratory phase is over, the process is called cycling. Different modes have different ways of cycling. These are: volume cycling [volume-controlled ventilation (VCV)], time cycling [pressure-controlled ventilation (PCV)] and flow cycling [pressure support ventilation (PSV)].

Q. What are the common or old modes of mechanical ventilation? What are the differences between intermittent mandatory ventilation (IMV), synchronized intermittent mandatory ventilation (SIMV) and assist control (AC)?

Ans. The standard or established modes are volume-controlled mode, pressure-controlled mode and pressure

support mode. The controlled modes can be further of following types: IMV, SIMV and AC. Sometimes, two modes may be combined during ventilation; for example, SIMV may be combined with pressure support. There are a wide variety of different modes available, many of which are proprietary and ventilator-specific.

In IMV, the ventilator delivers the set number of breaths at set time, regardless of which phase of respiration the patient is in. In SIMV, the inspiration is synchronized with patient effort, but the ventilator will deliver only fixed number of breaths; whereas in AC, the ventilator will deliver a set number of breaths synchronized with patient effort and will also assist any extra breaths (to the level of set tidal volume) that the patient may initiate.

Q. Discuss VCV. What are the advantages and disadvantages?

Ans. Volume-controlled ventilation is a controlled mode where the tidal volume is controlled and it also allows the patient to breathe additional breaths. This can also be used in SIMV or AC mode:

- *Changeover from expiration to inspiration:* This can be pressure- or flow-triggered, and each breath will have the same set tidal volume.
- *Inspiratory phase:* During VCV, the settings are tidal volume to be delivered each breath, PIFR usually 1–1.5 L/min, and inspiratory time. The combination of PIFR and the inspiratory time will determine how fast the breath will be delivered by the ventilator, how much inspiratory pause will be available and the time available for expiration. A very high inspiratory flow rate leads to high airway pressures, and a low inspiratory flow rate may lead to ventilator dyssynchrony and air hunger. The length of inspiration depends on the tidal volume and inspiratory flow rate that have been set. The flow during this mode is constant flow (square wave pattern).
Limit: The flow rate rises to the set limit (set flow rate), or the delivered tidal volume; provided the limit is not exceeded. In VCV, the limiting variable is pressure—so the modern VCV mode is volume-controlled pressure-limited mode. The limit prevents delivery of excessive pressures to the lungs which means that the preset tidal volume will not be delivered as well. So the limit setting is very important to prevent barotrauma.
- *Changeover from inspiration to expiration or cycling:* This mode is volume-cycled, which means that the ventilator cycles to exhalation when the set tidal volume has been delivered.
- *Expiratory phase:* Positive end-expiratory pressure may be added.

Advantages: VCV ensures delivery of set minimum tidal and minute ventilation regardless of patient effort and allows

the patient to rest by reducing work of breathing. Patient can however increase the minute ventilation by breathing spontaneously; however, the spontaneous breaths will not be supported.

Disadvantages: Airway pressure varies as per compliance of patient lungs. High inspiratory flow demands from patient may not be met as the PIFR is fixed. During this mode, adverse hemodynamic effects may occur due to positive intrathoracic pressure. Inappropriate hyperventilation may occur if the patient takes additional breaths above the set rate.

Q. Discuss PCV with advantages and disadvantages.

Ans. In this mode, inspiratory airway pressure and inspiratory time are fixed by the clinician:

- *Changeover from expiration to inspiration:* Inspiration is time-triggered with PCV, and the respiratory rate is set. When used as P-SIMV or PCV-AC patient may initiate additional breaths.
- *Inspiratory phase:* The peak pressure (P_{peak}) is set. The ventilator quickly delivers inspiratory flow to reach the set pressure and maintains the flow to keep the pressure constant. Therefore, the flow type is called decelerating flow (decreasing flow). Thus, peak inspiratory pressure is generated quickly and maintained constant throughout inspiration. With a prolonged inspiratory time, the inspiratory flow rate may fall to zero before the end of inspiration. The tidal volume delivered depends on the set pressure and inspiratory time, and on patient factors such as respiratory mechanics, i.e., airway resistance and respiratory compliance. Tidal volume may vary but the peak airway pressure is constant.
Limit: Gas flow occurs till the preset airway pressure is reached. This pressure is maintained constant throughout the set inspiratory time.
- *Changeover from inspiration to expiration:* PCV is time-cycled, which means that the ventilator cycles into expiration when the set inspiratory time is over. A long inspiratory time will increase the tidal volume delivered.
- *Expiratory phase:* Expiration occurs passively and PEEP can be added to pressure control ventilation.

Advantages: PCV ensures a control over peak airway pressure and allows the patient to rest by reducing work of breathing. Minimum minute ventilation is not ensured. This mode may be of some use in patients with small leaks (such as bronchopleural fistula) to tide over the initial crisis, till definite treatment can be undertaken. The decelerating flow may compensate for small leaks.

Disadvantages: Tidal volume (and minute ventilation) varies as per compliance of patient lungs and minimum minute ventilation is not assured. Inappropriate hyperventilation

may occur if the patient takes additional breaths above the set rate.

Q. Discuss PSV with advantages and disadvantages.

Ans. Pressure support is a spontaneous mode of ventilation. The patient initiates each breath, the ventilatory flow takes the inspiratory pressure to the set level and the patient terminates the inspiration. Apnea backup ventilation must be set while using this mode:

- *Changeover from expiration to inspiration:* The patient triggers (pressure or flow) each breath in PSV.
- *Inspiratory phase:* The clinician sets the inspiratory pressure. The ventilator delivers a high initial flow rate, to achieve set pressure, and then the flow declines to maintain the set airway pressure constant throughout inspiration. The idea is to set the support pressure so that the patient generates adequate tidal volumes with a comfortable respiratory rate.

Limit: Gas flow occurs till the set airway pressure is reached. This pressure is maintained constant throughout the inspiratory time.

- *Changeover from inspiration to expiration:* Pressure support is flow-cycled. As the patient's inspiratory flow rate falls to 25% of the peak inspiratory flow, the ventilator cycles into the expiratory phase.
- *Expiratory phase:* PEEP can be combined with pressure support.

Advantages: PSV is supposed to ensure patient-ventilator synchrony, reduce work of breathing and thus maintain patient comfort.

Disadvantages: If the patient stops breathing apnea backup is the only ventilation. The tidal volume and minute ventilation will vary as per patient's lung compliance. Presence of leaks in the ventilator circuit will interfere with cycling.

Q. Summarize the differences between VCV and PCV.

Ans. The **Table 1** summarizes the differences between VCV and PCV.

TABLE 1: Differences between volume-controlled ventilation (VCV) and pressure-controlled ventilation (PCV).

Variable	Volume/flow control	Pressure control
Tidal volume	Set, constant	Variable
Peak inspiratory pressure	Variable	Set, constant
Inspiratory time	Set	Set
Inspiratory flow	Set	Variable
Inspiratory flow waveform	Set, constant	Decelerating type, variable

Q. What is PEEP?

Ans. Positive end-expiratory pressure refers to a constant baseline pressure that is present throughout the ventilatory

cycle (not just on expiration). CPAP is PEEP during spontaneous breathing.

Q. How does PEEP improve oxygenation?

Ans. Positive end-expiratory pressure improves oxygenation by preventing end-expiratory collapse of alveoli and by recruiting nonventilated (shunt), or poorly ventilated (low ventilation-to-perfusion (V/Q)) alveoli. Cyclic opening and closing of alveoli and distal bronchioles with each breath during ventilation, depletes surfactant and contributes to ventilator-induced lung injury (VILI). PEEP keeps the alveoli inflated and prevents cyclic opening and closing of alveoli. PEEP improves oxygenation in patients with pulmonary edema by creating hydrostatic forces which move fluid from the alveoli and airways into the interstitium and by reducing left ventricular afterload.

Q. What are the adverse effects of PEEP?

Ans. Inappropriate PEEP may create overinflation of alveoli leading to lung injury. In patients with hypovolemia and high dead space [chronic obstructive pulmonary disease (COPD) patients], PEEP may worsen ventilation due to an increase in dead space. PEEP may worsen right heart failure by increasing right ventricular afterload. PEEP may precipitate hypotension due to raised intrathoracic pressure.

Q. What is intrinsic PEEP or auto-PEEP? How do you detect it?

Ans. Intrinsic PEEP is PEEP generated within patient's own lungs and is distinct from externally applied PEEP. It develops when expiratory time is short and expiration remains incomplete. Thus, when the next breath comes in, some amount of previous tidal volume is still present in the alveoli, thus the next ventilator breath "stacks" on the previous breath. This leads to hyperinflation of the alveolus and generation of positive pressure, this intrinsic increase in end-expiratory pressure known as "intrinsic or auto-PEEP". This process is seen commonly in COPD patients is called dynamic hyperinflation.

Ways to detect auto-PEEP: Many ventilators will permit direct measurement of auto-PEEP by doing an expiratory hold maneuver.

Clinical detection: Development of auto-PEEP should be anticipated in patients with a high minute ventilation, high respiratory rate, short expiratory times, and airway obstruction. Since auto-PEEP can cause severe hyperinflation, patient has to spend more effort; this is seen clinically patients failing to trigger ventilator or patient-ventilator dyssynchrony. If expiratory sounds are heard throughout the expiratory phase on auscultation right up to the inspiratory phase of the next breath then auto-PEEP should be suspected.

From flow waveform: Look at the expiratory flow versus time curve on the ventilator. If expiratory flow does not return to baseline (i.e., zero) before the next inspiration, then auto-PEEP is present.

Q. What are the clinical implications of auto-PEEP?

Ans. Auto-PEEP causes an increase in intrathoracic pressure and therefore can cause adverse hemodynamic effects. Auto-PEEP can increase the work of breathing required to trigger the ventilator. This additional imposed work of breathing can be very uncomfortable and will eventually lead to patient fatigue.

Q. How do you manage auto-PEEP?

Ans. Auto-PEEP is due to short expiratory time and is managed by increasing the expiratory time on the ventilator. This is achieved by reducing respiratory rate, reducing tidal volume or increasing the PIFR. In patients with bronchial asthma, apart from these changes to ventilatory setting, bronchodilator therapy will reduce auto-PEEP.

Q. What are the contraindications to PEEP?

Ans. Relative contraindications to the use of PEEP include:

- Hypotension due to volume depletion and dehydration.
- Right ventricular failure since PEEP increases right ventricular afterload.
- Right to left intracardiac shunt.
- Increased intracranial pressure (ICP) particularly if CVP is also high. Since hypoxemia worsens neuronal damage, risk of increased ICP with PEEP must be balanced with likelihood of an improvement in oxygenation.
- Patients with hyperinflation such as emphysema and COPD. However, external PEEP can be used to balance auto-PEEP in spontaneously breathing patients as it makes triggering easier for these patients.
- Bronchopleural fistula. High levels of PEEP may increase air leak with loss of ventilation. PEEP may also prevent healing.

Q. Discuss initial setup of the ventilator for elective ventilation of a postoperative patient.

Ans. First we have to select a mode. There are studies to suggest a difference in outcomes if either VCV or PCV is used. Select a mode that you are most familiar with. Since the patient is going to start breathing soon, an AC mode is useful as it will also assist all spontaneous breaths. The initial settings thus are as follows:

- *Fraction of inspired oxygen (FiO_2):* 1.0 always starts with 100% O_2 . This should be reduced to 0.5 as soon as possible, preferably within next 24 hours.
- *Tidal volume:* 8–10 mL/kg body weight.
- *Respiratory rate:* 12–14 breaths/minute.

- *PEEP:* Start with 5 cmH₂O, may be increased in increments of 2–3 cmH₂O for achieving adequate oxygenation.
- *Trigger sensitivity:* 1–3 L/min or –1 to –2 cmH₂O.
- *Inspiratory flow rate:* 1–1.5 L/kg, higher rates may be needed for asthma patients.
- The settings may be adjusted after assessing patient response and an arterial blood gas drawn 30 minutes later.

Q. What alarms are set during ventilation? Explain the function of each alarm.

Ans. The most common set alarms are:

- *High pressure alarm:* Sets off when the compliance is bad or patient coughs or when there are with mechanical problem with circuit.
- *Low pressure alarm:* Sets off with leaks in the circuit, accidental disconnection, or accidental extubation.
- *Low minute ventilation:* Sets off with leaks in the circuit or inappropriate settings.
- *High minute ventilation:* When the patient is breathing too fast, due to need for high MV or if patient not sedated well enough.
- High or low respiratory rate.
- High and low FiO_2 .
- *Apnea alarm:* circuit disconnection, apnea (during spontaneous modes). When using spontaneous modes, backup ventilation must be set up.

Q. What are the complications of mechanical ventilation?

Ans.

Related to Intubation

- Endobronchial intubation and massive atelectasis
- Esophageal intubation
- Failed intubation
- Trauma to upper airway and tracheobronchial tree
- Blocked tracheal tube
- Accidental extubation
- Sinusitis
- Cuff leak
- Tracheal stenosis
- Tracheoesophageal fistula
- Tracheoinnominate fistula.

Due to Positive Intrathoracic Pressure

- Hypotension
- Reduced organ perfusion
- Raised ICP
- Ventilator malfunction
- Meteorism [gaseous distention of gastrointestinal (GI) tract]
- Ventilator-induced lung injury or ventilator-associated lung injury (VALI).

Suction-Related Complications

- Hypoxia
- Arrhythmias.

Related to Ventilator Setup

- Unintended hyperventilation
- Unintended hypoventilation (acute respiratory acidosis)
- Ventilator-initiated lung injury
- Inadvertent air trapping and auto-PEEP
- Respiratory distress (due to patient ventilator asynchrony—"fighting the ventilator")
- Excessive patient work of breathing (due to incorrect setting)
- Inappropriate use of paralytic agents
- Oxygen toxicity
- Ventilator-associated pneumonia.

Technology Gap in Patient Management

- Bedside communication failure
- Muscle weakness
- Barotrauma
- Subcutaneous emphysema
- Pneumomediastinum
- Pneumothorax.

Q. What is barotrauma?

Ans. Pneumothorax, pneumomediastinum, and subcutaneous emphysema are manifestations of extra-alveolar air, and are collectively termed "barotrauma" in mechanically ventilated patients, although in most instances alveolar overdistention (high peak inflation volume) is probably responsible for their occurrence rather than high airway pressure. The occurrence of alveolar rupture during mechanical ventilation is influenced markedly by the presence and nature of underlying lung pathology. Barotrauma is less common in patients with normal lungs, and more common in patients with severe obstructive lung disease as well as in acute respiratory distress syndrome (ARDS).

Manifestations of Barotrauma

- Pulmonary interstitial emphysema
- Pneumomediastinum
- Subcutaneous emphysema
- Pneumopericardium
- Systemic air embolism
- *Pneumothorax:* It may be asymptomatic, or present with worsening oxygenation, an increase in peak inspiratory airway pressure, or a fall in lung-chest wall compliance. Tension pneumothorax is defined by its adverse effects on cardiovascular function than by its radiographic characteristics. The latter include mediastinal displacement away from the pneumothorax, reversal

of the diaphragmatic curve, and overall enlargement of the affected hemithorax. Most commonly the patient with tension pneumothorax presents with agitation and respiratory distress, hypotension and other signs of cardiovascular collapse.

Q. What is VILI? What are the mechanisms which lead to VILI?

Ans. Injury to the lung that occurs in the course of and due to mechanical ventilation is termed ventilator-induced lung injury. The following mechanisms of lung injury have been described:

- *Atelectrauma:* Cyclical collapse of alveoli, i.e., repeated opening and closing of alveoli and surfactant depletion. Normal or stable alveoli do not collapse completely at the end of expiration. During ARDS, some alveoli may become unstable and undergo cyclical collapse. Ventilation with high FiO_2 aggravates alveolar collapse due to absorption atelectasis.
- *Oxygen toxicity:* While this is well-known, it is not clear what concentration of oxygen is toxic over what period of time. It is generally assumed that $\text{FiO}_2 < 0.6$ is not toxic; however, an attempt must be made to maintain the FiO_2 as low as possible.
- *Volutrauma:* Ventilation at high volumes and pressures can lead to alveolar overdistention, causing increased permeability in the uninjured lung with pulmonary edema and enhanced edema in the injured lung.
- *Cyclical shear stress injury:* Cyclic opening and closing of atelectatic alveoli during mechanical ventilation create tremendous shear stress at their junctions with open alveoli. This results in damage to the capillary endothelium and the alveolar membrane.
- *Biotrauma:* Alveolar overdistention along with the repeated collapse and reopening of the alveoli can result in a whole cascade of proinflammatory cytokines which induce a pulmonary and systemic cytokine response, aggravating lung injury and causing systemic multiorgan dysfunction.
- *Barotrauma:* Pneumothorax, pneumomediastinum, interstitial emphysema.

Q. How can we monitor the mechanics of the respiratory system during mechanical ventilation? Explain the terms peak pressure and plateau pressure.

Ans. The respiratory mechanics can be monitored by looking at the pressure-time graph present on all modern ventilators (**Fig. 2**). The highest pressure during inspiration in a volume-controlled mode is peak pressure or Ppeak.

When you apply an inspiratory pause, the alveoli are held distended with no gases flowing in or out of the lungs. This pressure is called alveolar distending pressure

of plateau pressure or Pplat. In patients with alveolar pathologies, this pressure will go up.

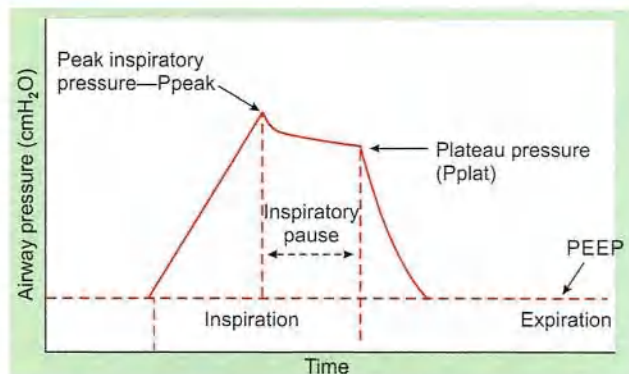


Fig. 2: Pressure-time graphic used to monitor respiratory mechanics.

Q. What is the compliance of the respiratory system? How do you measure static compliance (Cstat) and dynamic compliance (Cdyn)?

Ans. Compliance relates to the change in volume of the system on application of pressure. Cstat is measured when there is no movement of gases occurring in the system (Fig. 2), i.e., alveoli are held open at the end of inspiration. So Pplat is a surrogate of Cstat.

Static compliance is given by the following formula: $C_{stat} = \text{Tidal volume} / (P_{plat} - PEEP)$.

Alveolar pathologies such as pneumonia or ARDS will cause a decrease in Cstat and an increase in Pplat. Cdyn is measured when gas is flowing into the lung. This means the flow has to overcome the resistance of the breathing system starting with inspiratory limb of the circuit, endotracheal tube and the tracheobronchial tree. Thus at the level of Ppeak, if there is anything wrong with the inspiratory arc of the circuit—such as a linked ET tube, bronchospasm, it will lead to requirement of increased inspiratory pressures to overcome the resistance.

Dynamic compliance is given by the following formula: $C_{dyn} = \text{Tidal volume} / (P_{peak} - PEEP)$.

Peak pressure is a surrogate of Cdyn. The effect of therapy in a patient with bronchospasm can be monitored by tracking a decrease in Ppeak or Cdyn.

Q. How is resistance of the respiratory system calculated?

Ans. Resistance of any system is given by the formula $\text{resistance} = \text{Pressure across the tubing} / \text{Flow in the system}$. For respiratory system, this will be given by $\text{resistance} = (P_{peak} - P_{plat}) / \text{Inspiratory flow}$ (Fig. 2). At the bedside, the difference between Ppeak and Pplat can be easily monitored as a surrogate of the calculated resistance. If Ppeak is high but Pplat is normal, the patient has a resistive problem. Once you start treating the bronchospasm, this difference

will reduce. In a patient with alveolar or lung parenchymal pathology, both Ppeak and Pplat will be high and close together.

Q. What is bilevel inspiratory positive pressure ventilation (BiPAP)?

Ans. This is probably the most confusing term in the area of ventilation. Theoretically, all pressure-limited modes can be called bilevel positive airway pressure (BiPAP) as they work between two pressures: (1) the inspiratory pressure and (2) the expiratory pressure. The various types of BiPAP are described here:

- **BiPAP type 1 (BiPAP, Respironics):** This is completely spontaneous mode. The clinician sets the inspiratory positive airway pressure (IPAP) and an expiratory positive airway pressure (EPAP). The patient triggers the breath, ventilator supplies a decelerating flow, the breath is limited by the IPAP. Cycling is flow sensed and the expiration then ends at the EPAP pressure level. Thus, it is similar to a CPAP + PS mode where IPAP is the PS level and CPAP is EPAP.
- **BiPAP type 2 (BiPAP, Drager):** This is a controlled mode of ventilation. The clinician sets the IPAP and an EPAP, inspiratory time and expiratory time. The ventilator triggers the breath which is pressure-controlled, time-cycled and ends at the set EPAP level.
- **BiPAP type 3 (Drager Evita):** This mode is similar to BiPAP type 2 and the settings are similar except that the time settings at IPAP and EPAP are set longer (5–10 seconds each) and the patient can breathe spontaneously. Thus, this can be considered to be a double CPAP mode, where patient breathes alternately between two levels of pressure (IPAP and EPAP).
- **BiPAP type 4:** This is a variation of type 3 breath. The settings are similar to the type 3 mode above, except that the IPAP time is too short. The patient can breathe spontaneously at the lower pressure level, i.e., CPAP but not at the IPAP level. Thus, the inspiration is a pressure-limited, time-cycled breath. This is similar to a pressure-limited and time-cycled form of SIMV. Additional breaths are spontaneous unsupported breaths or they may be pressure supported breaths.
- **BiPAP type 5:** This is another variation on type 3 BiPAP. Here the patient breathes spontaneously only at the IPAP level. The expiratory time is very short and does not allow spontaneous breathing.

Q. What is pressure control inverse ratio ventilation (PC-IRV)?

Ans. Pressure control inverse ratio ventilation is a type of pressure control continuous mandatory ventilation (CMV) where all breaths are pressure-limited and time-cycled and ventilator-triggered. The inspiration is longer than the

expiration. It is also simply called inverse ratio ventilation. In patients with ARDS, the alveoli in various lung regions may have different time constants, i.e., some alveoli are “slow” while some are “fast”; this basically means that these alveoli are slow (or fast) to fill or empty. When we use inverse ratio ventilation, the slower alveoli get time to be filled.

Q. Describe airway pressure release ventilation (APRV).

Ans. It is actually a variation of BiPAP one. Actually, this is pressure-controlled mode with inverse ratio ventilation. It is often described as two levels of positive pressure applied for preset time between these pressure levels to breathe spontaneously. Because the patient breathes between two positive pressures, this has also been called bilevel positive airway pressure. Other synonyms are variable positive airway pressure (VPAP), intermittent CPAP and CPAP with release. Here the mandatory breaths are pressure-controlled, time-triggered, pressure-limited and time-cycled. APRV allows the patient to breathe spontaneously during any phase of ventilators’ mechanical cycle. The clinician sets the pressure high (generally the P_{plat}) and the pressure low or release pressure (generally set as 0) and the time spent at each pressure. Thus, both time high (generally 5–6 seconds) or inspiratory time and time low or time for expiration (generally 0.5–1 second) are also set. The expiratory time is set such that the expiration is incomplete. Thus, oxygenation improves because of prolonged inspiratory time (helps recruit and ventilate the slow alveoli) and development of auto-PEEP due to incomplete expiration.

Q. Describe high-frequency ventilation.

Ans. This is an infrequently used mode of ventilation. The ventilators used are totally different and the settings also are different from the conventional ventilators. Here one uses very small tidal volumes and very high frequencies (180–300 Hz). The tidal volumes are generally less than the anatomical dead space. All breaths are ventilator-triggered and time-cycled. A recent review described mechanism of gas exchange that can be briefly summarized as convection and convection-diffusion. The diffusion occurs due to turbulent movement of gases and direct ventilation of close alveoli. Due to different patterns of inspiratory and expiratory velocity profiles of the tidal ventilation, axial movement of gas flow occurs leading to gas exchange with the expired gas. Other mechanisms include pendelluft, cardiogenic mixing, laminar flow with Taylor dispersion, collateral ventilation between neighboring alveoli, and molecular diffusion takes place. This mode has mostly been shown to be effective in management of ARDS in neonates. It can also be used during surgery of the airway. Disappointingly, the studies of high-frequency oscillatory ventilation (HFOV) in adult patients in ARDS have failed to show benefit.

Q. What are dual modes of ventilation? How do these modes work?

Ans. Dual control modes are capable of controlling either pressure or volume based on a measured input variable. These modes switch between pressure control and volume control ventilation. This switch can occur either within single breath, e.g., volume-assured pressure support (VAPS) ventilation and pressure augmentation (PA) or the switch can occur between two breaths: volume support (VS) or pressure-regulated volume control (PRVC). These modes are also called closed-loop modes of ventilation, i.e., as per the sensed change in requirements of patient; the ventilator changes the delivery of breath. In theory, closed-loop ventilation should lead to increased safety to the patient, greater patient comfort and by virtue of their very definition show a rapid response to change in patient condition. Unfortunately, the introduction of these modes has not been shown to contribute to reduced length of mechanical ventilation or improved intensive care unit (ICU) outcomes.

Dual control breath to breath: These modes are essentially pressure-limited modes that aim to maintain minimum P_{peak} to achieve the set tidal volume. The tidal volume is maintained consistently in face of varying compliance or resistance, thus we get an automated reduction of pressure and flow while constant minute volume is maintained. The examples include PRVC and VS.

Dual control within a breath: In this type of ventilation, the modes aim to maintain minimum P_{peak} required to achieve the tidal volume set by the clinician (a more subtle volume control mode). The tidal volume is maintained in face of varying compliance or resistance by automatic reduction or increase of pressure and flow and thus eventually maintaining the desired minute volume.

Q. What is neurally adjusted ventilatory assist (NAVA)? How does it work?

Ans. The neural signal of respiration originates in respiratory center which is then transmitted through phrenic nerve to excite the diaphragm. In the novel mode called neurally adjusted ventilatory assist, a nasogastric tube containing seven electrodes is placed in the stomach so that the electrodes are at the level of diaphragm. The signals coming to diaphragm from the respiratory center are sensed by these electrodes and the information is sent to the ventilator. The ventilator then provides assistance to the patient commensurate with the degree of patient effort. Thus with this mode patient’s own respiratory center is in direct control of the mechanical support required on a breath-by-breath basis, and any variation in neural respiratory demand is responded to by the appropriate corresponding change in ventilatory assistance. So in theory NAVA should

be very useful in reducing patient ventilator asynchrony. This mode is relatively new and clinical studies are ongoing regarding utility of this mode.

Q. What is automatic tube compensation (ATC)? How does it work?

Ans. Automatic tube compensation is a new option which can be used with other modes of ventilation. During inspiration and expiration, it can compensate for the nonlinear decrease in pressure through the endotracheal or tracheostomy tube. ATC compensates for the tube-related additional work of breathing. This is also sometimes termed the "electronic extubation". If the clinician thinks patient is ready for extubation, he can put the ATC on. If the patient appears to breathe he may be extubated. However, patient can still fail extubation due to upper airway edema.

■ SUGGESTED READING

1. Brochard L, Martin GS, Blanch L, Pelosi P, Belda FJ, Jubran A, et al. Clinical review: respiratory monitoring in the ICU—a consensus of 16. *Crit Care*. 2012;16:219.
2. Cohen JD, Shapiro M, Grozovski E, Lev S, Fisher H, Singer P. Extubation outcome following a spontaneous breathing

trial with automatic tube compensation versus continuous positive airway pressure. *Crit Care Med*. 2006;34:682-6.

3. Coisel Y, Chanques G, Jung B, Constantin JM, Capdevila X, Matecki S, et al. Neurally adjusted ventilatory assist in critically ill postoperative patients: a crossover randomized study. *Anesthesiology*. 2010;113:925-35.
4. Gould T, de Beer JM. Principles of artificial ventilation. *Anaesth Intensive Care Med*. 2007;8:91-101.
5. Guttman J, Haberthür C, Mols G, Lichtwarck-Aschoff M. Automatic tube compensation (ATC). *Minerva Anesthesiol*. 2002;68:369-77.
6. Habashi NM. Other approaches to open-lung ventilation: airway pressure release ventilation. *Crit Care Med*. 2005;33:S228-40.
7. Kulkarni AP, Divatia J. Ventilation-induced lung injury. In: Nayyar V, Srinivas S, Kishen R, Peters JV (Eds). *Critical Care Update*. New Delhi: Jaypee Brothers Medical Publishers (P) Ltd; 2009.
8. MacIntyre NR, Branson RD. *Mechanical Ventilation*, 2nd edition. Philadelphia: WB Saunders Company; 2009.
9. Pillow JJ. High-frequency oscillatory ventilation: mechanisms of gas exchange and lung mechanics. *Crit Care Med*. 2005;33:S135-41.
10. Tobin MJ. Advances in mechanical ventilation. *N Engl J Med*. 2001;344:1986-96.

Pulmonary Function Tests

Vijaya Prakash Patil, Madhavi Desai, Debashree Lahiri

Q. What are pulmonary function tests?

Ans. “Pulmonary function tests (PFTs)” is a term used to indicate a group of studies or maneuvers that may be performed using standardized equipment to measure lung function. PFTs can include simple screening spirometry, formal lung volume measurement, diffusing capacity for carbon monoxide, and arterial blood gases (ABGs). These studies may collectively be referred to as a complete pulmonary function survey. It includes:

- Spirometry
- Lung volumes by helium dilution or body plethysmography
- Diffusing capacity for carbon monoxide
- Bronchial challenge testing
- *Blood gases:* This is discussed in a separate chapter.
- Exercise tests
- Pulse oximetry.

Pulmonary function tests identify and quantify many functional and structural abnormalities of respiratory system. PFTs give information regarding the volume and velocity of the movement of air moving in and out of lungs, compliance of the lungs and chest wall, diffusion characteristics of membrane, and quantify the response to a procedure (lobectomy/pneumonectomy) and therapy. They only support or exclude a diagnosis. A combination of a thorough history and physical examination, as well as supporting laboratory data and imaging will help establish a confirmed diagnosis.

Q. What are the limitations of PFTs?

Ans.

- There is some variability in normal predictive value as with many other tests.
- The accuracy of the test depends on well-trained technician as well as patient's cooperation unlike other laboratory tests. Patient should use maximum efforts

and technician should be able to recognize submaximal efforts.

- Pulmonary function tests must be interpreted in the context of a proper history, physical examination, and ancillary diagnostic tests. Used alone they generally cannot distinguish among the potential causes of the abnormalities.

Q. What are the requirements for good PFTs?

Ans. The American Thoracic Society (ATS) has published guidelines for the standardization of spirometry equipment and performance which include criteria for acceptability and reproducibility. Because spirometry is such an effort dependent test, each spirogram should be examined using performance criteria set forth by the ATS:

- Lack of artifact induced by coughing, glottic closure, or equipment problems (primarily leak)
- Satisfactory start to the test without hesitation
- Satisfactory exhalation with 6 seconds of smooth continuous exhalation and/or a plateau in the volume time curve of at least 1 second or a reasonable duration of exhalation with a plateau.

Criteria for reproducibility after obtaining three acceptable spiograms include:

- Largest forced vital capacity (FVC) within 0.2 L of next largest FVC
- Largest forced expiratory volume in 1 second (FEV1) within 0.2 L of next largest FEV1.

If the two above criteria have not been met, additional spiograms should be obtained.

Up to eight efforts should be allowed in order to meet acceptability and reproducibility criteria. Beyond eight efforts, fatigue may play a role in the results and the interpretation of the test may not be reliable. If all the criteria are not met, abnormal results should be interpreted with caution.

Q. What are the indications for spirometry?

Ans.

Diagnostic

- To evaluate symptoms, signs or abnormal laboratory tests:
 - *Symptoms:* Dyspnea, wheezing, orthopnea, cough, phlegm production, chest pain
 - *Signs:* Diminished breath sounds, overinflation, expiratory slowing, cyanosis, chest deformity, unexplained crackles
 - *Abnormal laboratory tests:* Hypoxemia, hypercapnia, polycythemia, abnormal chest radiographs
- To measure the effect of disease on pulmonary function—obstructive or restrictive
- To screen individuals at risk of having pulmonary diseases:
 - Smokers
 - Individuals in occupations with exposures to injurious substances affecting lungs, e.g., asbestosis
- To assess preoperative risk—this is especially true of patients who are:
 - Known to have pulmonary disease
 - Obese
 - Undergoing an upper abdominal or a thoracic operation
 - Have a history of smoking, cough or wheezing
- Part of routine physical examinations
- To assess prognosis (idiopathic pulmonary fibrosis, lung transplant, etc.)
- To assess health status before enrollment in strenuous physical activity programs.

Monitoring

- To assess therapeutic interventions:
 - Bronchodilator therapy
 - Steroid treatment for asthma, interstitial lung disease, etc.
 - Other (antibiotics in cystic fibrosis, etc.)
- To describe the course of diseases affecting lung function:
 - Pulmonary disease
 - Obstructive airway disease
 - Interstitial lung disease
 - Cardiac disease, congestive heart failure
 - Neuromuscular disease, Guillain-Barré syndrome
 - To monitor persons in occupations with exposure to injurious agents
 - To monitor for adverse reactions to drugs with known pulmonary toxicity (bleomycin, amiodarone, etc.).

Public Health

- Epidemiologic surveys
- Comparison of health status of populations living in different environments

- Validation of subjective complaints in occupational/environmental settings
- Derivation of reference equations for interpretation of PFTs for age, sex, race.

Q. Are there any contraindications for spirometry?

Ans. There are no absolute contraindications for performing PFTs. Relative contraindications for spirometry include:

- Hemoptysis of unknown origin
- Pneumothorax
- Recent myocardial infarction
- Unstable angina pectoris
- Thoracic, abdominal and cerebral aneurysms
- Recent abdominal or thoracic surgical procedures
- Patients with a history of syncope associated with forced exhalation
- Recent eye surgery (raised intraocular pressure during forced expiration).

Q. What is spirometry?

Ans. Spirometry is one of the most commonly performed PFT which measures the rate at which the lungs change volume during quiet and forced breathing maneuvers. It can only measure lung volume compartments that exchange gas with the atmosphere.

There are two methods of recording FVC:

1. Volume exhaled is plotted as a function of time, where we commonly measure FEV1 and the average forced expiratory flow (FEF) rate over the middle 50% of FVC, i.e., FEF 25–75% which is effort independent.
2. Volume exhaled is plotted against flow (flow-volume curve). Flow-volume loop is a plot of inspiratory and expiratory flow on the Y-axis against volume on the X-axis during the performance of maximally forced inspiratory and expiratory maneuvers.

Both the curves reflect same data and most of the machines that are used presently are computerized and give both curves.

Q. Enumerate physiological factors that affect normal values of PFTs.

Ans. Spirometry is typically reported in both absolute values and as a predicted percentage of normal. Standards for normality are based on age, sex, height, weight and race with an abnormal value occurring if there is a difference of $\pm 20\%$ from the predicted mean value:

- *Age:* Natural elasticity of the lungs decreases with aging which results in gradual decrease of lung volumes and capacities.
- *Gender:* Lung volumes and capacities of males are more than those of female individuals. Even when males and females are matched for height and weight, males have larger lungs than females. Because of this gender-

dependent lung size difference, different normal tables must be used for males and females.

- **Body height and size:** An individual with small stature has smaller PFT result than a man of the same age who is much larger. Normal tables account for this variable by giving predicted PFT data for males or females of a certain age and height. In obese patients, body fat to lean body mass ratio increases, the abdominal mass prevents downward excursion of the diaphragm and the PFT results will demonstrate a smaller measured PFT outcome than expected. This leads to smaller measured values than the predicted values (predicted values from the normal tables).
- **Race:** Caucasians PFTs are different than those of Blacks, Hispanics, and Native Americans.

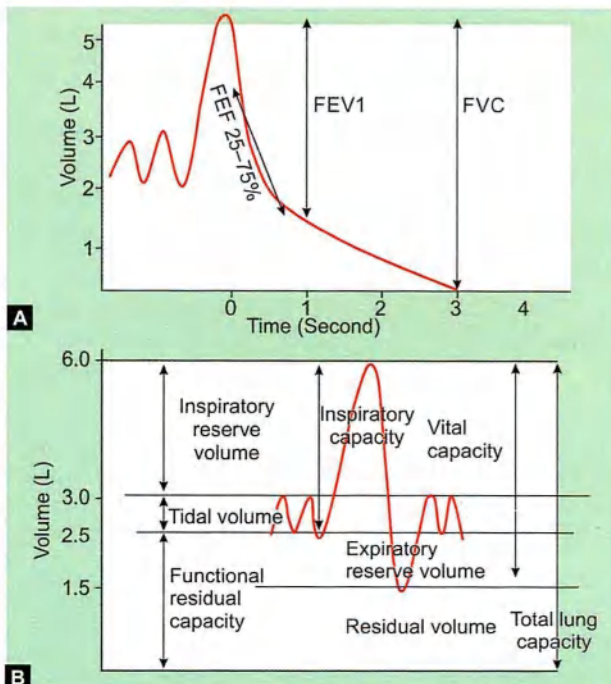
Q. How will you decide normal range of PFTs for a patient with kyphoscoliosis?

Ans. Body height should not be used in these patients as the measured height is less than actual skeletal height due to deformed spine. Measurement of arm span is better marker of actual height in these patients.

Q. Give definitions of various volumes and capacities that are mentioned in PFT reports.

Ans. Various volumes and capacities of PFT are shown in **Figures 1A and B:**

- Tidal volume (VT) is the volume of air that is inhaled or exhaled with each breath when a person is breathing at rest.



Figs. 1A and B: Various volumes and capacities of pulmonary function test (PFT).

- Forced expiratory volume in 1 second in liters is the volume of air forcibly expired from a maximum inspiratory effort in the first second.
- Functional residual capacity (FRC) in liters is the volume of air in the lungs following a VT exhalation = Expiratory reserve volume (ERV) + Residual volume (RV).
- Forced vital capacity in liters is the total volume that can be forcefully expired from a maximum inspiratory effort.
- Expiratory reserve volume in liters is the maximum volume of air that can be exhaled from the tidal end-expiratory position.
- Inspiratory capacity (IC) in liters is the maximum volume of air that can be inhaled from VT end-expiratory level; the sum of inspiratory reserve volume (IRV) and VT.
- Inspiratory reserve volume is the maximum volume of air that can be inhaled from the end-inspiratory tidal position.
- Peak expiratory flow (PEF) is the highest FEF (L/second).
- Residual volume is the volume of air that remains in the lungs after maximal exhalation.
- Total lung capacity (TLC) is the total volume of air in the lungs at full inhalation; the sum of all volume compartments (IC + FRC or IRV + VT + ERV + RV).
- Vital capacity is the maximum volume of air that can be exhaled starting from maximum inspiration.
- Total lung capacity can be measured either as slow vital capacity (SVC) or FVC.

Q. What do you understand by static and dynamic lung volumes?

Ans. Static lung volumes reflect the elastic properties of the lungs and chest wall. These tests include all the capacity measurements: vital capacity, FVC, TLC, and FRC. Dynamic volumes and capacities are based on time and these tests reflect the caliber and integrity of the airways, e.g., FVC, FEV₁, maximum voluntary ventilation (MVV), etc.

Q. What is a restrictive lung disease?

Ans. The test is most reliably interpreted as showing restrictive abnormality is on the basis of TLC. If the TLC is not available, one may interpret a reduction in the vital capacity without a reduction of the FEV₁/vital capacity ratio as a restriction of the volume excursion of the lung. The severity of the abnormality might be graded as follows:

- **Based on the TLC:**
 - **Mild:** Predicted TLC is less than lower limit of normal but $\geq 70\%$
 - **Moderate:** Predicted TLC $< 70\%$ and $\geq 60\%$
 - **Moderately severe:** Predicted TLC $< 60\%$
- **Based on spirometry:**
 - **Mild:** Predicted vital capacity is less than lower limit of normal but $\geq 70\%$
 - **Moderate:** Predicted vital capacity $< 70\%$ and $\geq 60\%$

- *Moderately severe*: Predicted vital capacity < 60% and $\geq$ 50%
- *Severe*: Predicted vital capacity < 50% and $\geq$ 34%
- *Very severe*: Predicted vital capacity < 34%.

Q. What are the causes of restrictive PFTs?

Ans. Restrictive lung volumes can be produced due to various factors that include lung pathology, pleural pathology, chest wall pathology or muscular problems.

- *Lung parenchyma pathology*:
 - Resection of lung parenchyma, e.g., prior pneumonectomy/lobectomy
 - Atelectasis
 - Fibrosis as in old healed Koch's/interstitial fibrosis
 - Pulmonary edema as in congestive cardiac failure
 - Thickened pleura
 - Large tumor—intraparenchymal/pleural/mediastinal
 - Emphysema
- *Interpleural pathology*:
 - Effusion
 - Cardiomegaly
 - Tumor
 - Displacement of chest wall/diaphragm
 - Ascites
 - Pregnancy
 - Severe pain leading to splinting
 - Obesity
 - Kyphoscoliosis
 - Severe burns with contracture to scleroderma
- *Neuromuscular problems*:
 - Phrenic nerve palsy—unilateral/bilateral
 - Any neuromuscular disorder involving thoracic muscles: Polio, myasthenia gravis, Guillain-Barré syndrome, etc.

Q. What are the causes of obstructive PFTs?

Ans. Flow through the tubular passage of airways of the lung can be reduced for various reasons:

- Narrowing of the airways due to bronchial smooth muscle contraction: Asthma
- Narrowing of the airways due to inflammation and swelling of bronchial mucosa and the hypertrophy and hyperplasia of bronchial glands: Bronchitis
- Material inside the bronchial passage physically obstructing the flow of air: Mucus plugs, foreign objects or invasive tumors
- Destruction of lung tissue with the loss of elasticity and hence the loss of the external support of the airways: Emphysema
- External compression of the airways by tumors and trauma.

Q. How do you grade severity of obstructive lung disease from spirometry values?

Ans. The ATS has recommended gradation of severity as follows:

- FEV1/FVC < predicted and FEV1 > 100% of predicted—normal
- FEV1/FVC < predicted and FEV1 < 100% > 70% of predicted—mild obstruction
- FEV1/FVC < predicted and FEV1 < 70% > 60% of predicted—moderate obstruction
- FEV1/FVC < predicted and FEV1 < 60% > 50% of predicted—moderately severe obstruction
- FEV1/FVC < predicted and FEV1 < 50% > 34% of predicted—severe obstruction
- FEV1/FVC < predicted and FEV1 < 34% of predicted—very severe obstruction.

Q. What are the criteria for reversibility of small airway obstruction on PFTs?

Ans. Patients showing small airway obstruction on PFT should be tested twice—once before bronchodilators, and then after administration of bronchodilators to evaluate the responsiveness to a bronchodilator medication. The drug almost always used is a beta-2 selective sympathomimetic because it causes bronchodilation but which does not stimulate the heart to any great degree. If two out of three measurements (FVC, FEV1 and FEF 25–75%) improve, then it can be said that the patient has a reversible airway obstruction that is responsive to medication:

- *FVC*: An increase of 10% or more
- *FEV1*: An increase of 200 mL or 15% of the baseline FEV1
- *FEF 25–75%*: An increase of 20% or more.

In patients with airway obstruction, absence of a response to a single exposure to a bronchodilator, however, does not preclude a beneficial response to maintenance therapy and many clinicians prefer to use bronchodilators even in the absence of above-mentioned criteria.

Q. What is maximum voluntary ventilation and its significance?

Ans. The subject is asked to breathe as hard and as fast as possible for 10–15 seconds and the value obtained is then converted to 60 seconds and reported as liters per minute. For example, if the patient's MVV is 30 L in 15 seconds, it will be reported as $30 \times 4 = 120$ L/m. This test reflects the status of the respiratory muscles, compliance of the chest wall and lung, and airway resistance. Since it parallels the FEV1, it can be used to test internal consistency and estimate patient cooperation. It is a quick and easy way to assess the strength of the patient's pulmonary musculature prior to surgery, a poor result on this test suggests that the patient may have pulmonary problems postoperatively.

due to muscle weakness. But this test is effort dependent, if patient does not perform the test properly; it cannot be then used to assess the true pulmonary strength and compliance. When the MVV is disproportionately low in a patient who seems to be cooperating, neuromuscular weakness should be suspected. Except in advanced neuromuscular disease, most patients can generate fairly good single-breath efforts (e.g., FVC). But since MVV is much more demanding, it can reveal diminished reserves of weak respiratory muscles. A low MVV can occur in any type of pulmonary disorder like obstructive, restrictive, or in patients with heart disease and very frail patients.

Q. What are different methods of measuring residual lung volume?

Ans.

- *Gas dilution techniques* use either closed-circuit helium dilution or open-circuit nitrogen washout:
 - *Nitrogen washout technique:* In this technique, at the end of a normal expiration, the patient breathes 100% oxygen and all the nitrogen in the lung is “washed out.” The exhaled volume and the nitrogen concentration in that volume are measured. The difference in nitrogen volume at the initial concentration and at the final exhaled concentration allows a calculation of intrathoracic volume, usually FRC.
 - *Helium dilution technique:* It is based on the inhalation of a known concentration and volume of an inert tracer gas, such as helium, followed by equilibration of 7–10 minutes in the closed-circuit. The final exhaled helium concentration is diluted in proportion to the unknown volume of air in the patient’s chest (RV). Usually, the patient is connected at the end-tidal position of the spirometer; therefore, the lung volume measured is FRC.
- *Body plethysmography:* It is based on the principle of Boyle’s law, which states that the volume of gas at a constant temperature varies inversely with the pressure applied to it. In this technique, the patient sits in a closed “body box” with a known volume and the lung volumes are calculated based on the amount of air displaced from the box during ventilation.

Q. What is the significance of vital capacity?

Ans. Vital capacity reflects the patient’s ability to take a deep breath, to cough, and to clear the airways of excess secretions, therefore, is an important preoperative assessment tool. It is normally 70 mL/kg ideal body weight.

- Significant reductions in vital capacity < 20 cc/kg of ideal body weight indicate that the patient is at a higher risk for postoperative respiratory complications.

- Evaluating the patient’s condition for weaning from a ventilator. If the patient on a ventilator can demonstrate a vital capacity of 10–15 mL/kg of body weight, it is generally considered that there is enough ventilatory reserve to try weaning and extubation.

Q. What is the significance of residual volume?

Ans. Residual volume is the air that is remaining in lung at the end of forced expiration. It depends on two factors—limits of chest wall excursion and smaller airway collapse. In restrictive lung pathologies, RV is determined by chest wall compression by muscles. In obstructive diseases, the collapse of terminal airways prevents distal air from escaping the lungs and thus influences RV. RV and thus TLC are commonly increased in chronic obstructive pulmonary disease (COPD) patients but are normal in patients with asthma.

Q. What is the significance of FRC?

Ans. Functional residual capacity is the lung volume at which the inward elastic recoil of lungs is balanced by outward elastic force of relaxed chest wall. It is usually 40–50% of TLC (30 mL/kg ideal body weight). FRC increases with reduced lung elasticity and decreases with increased lung recoil. It is also affected by posture. FRC is more in standing than supine position and this is one of the reasons why during postoperative recovery patients should be kept in head elevated position. Almost all anesthetics reduce muscle tone, which in turn, lower FRC to close to the RV in awake state. This is the cause of faster desaturation of obese, pregnant patients or patients with large intra-abdominal mass under anesthesia. FRC increases with age because of loss of elastic lung tissue. The preoxygenation prior to induction of anesthesia is to replace the FRC with 100% O₂ which helps in delaying the desaturation of apneic patient during intubation.

Q. What is Gestalt approach of spirometry interpretation?

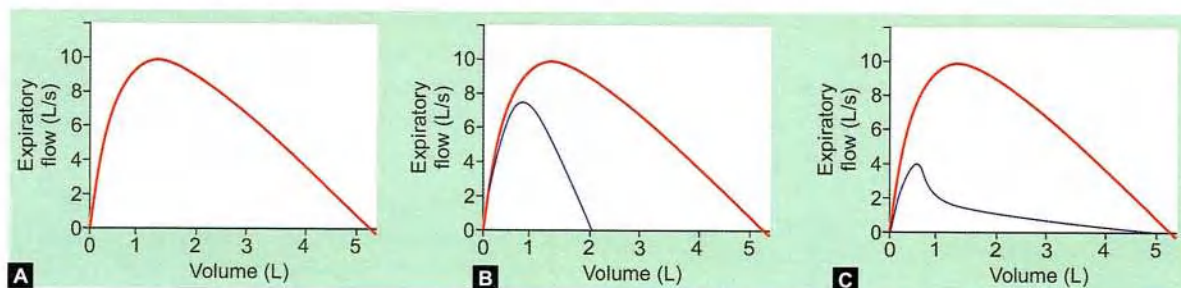
Ans. Here the patient’s flow-volume curve is superimposed with the predicted normal flow-volume curve for that patient so that instead of looking at numbers one can visually compare both curves. The difference in area provides a visual index of ventilatory limitation (**Figs. 2A to C**).

Q. Looking at PFTs can you make out which are good and which are unaccepted maneuvers?

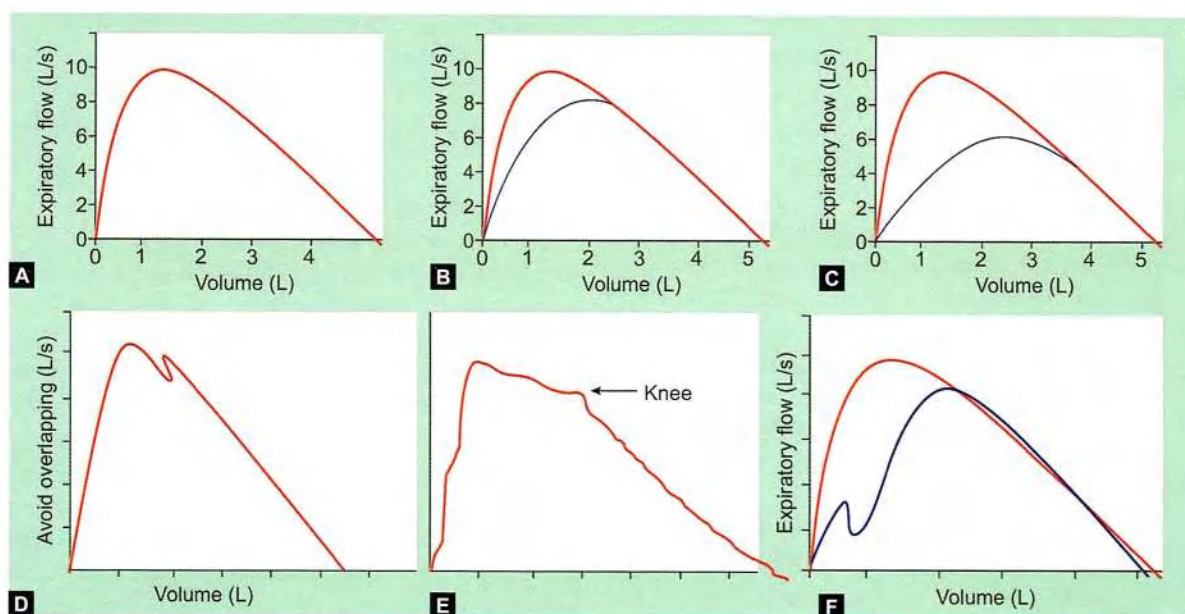
Ans. Good and acceptable maneuvers of PFTs are shown in **Figures 3A to F**.

Q. What is slow vital capacity test?

Ans. This test is performed by asking the patient to slowly and completely blow out all the air from his lungs. The SVC test eliminates the strong bronchoconstriction that



Figs. 2A to C: Gestalt approach of spirometry interpretation.



Figs. 3A to F: (A) Excellent efforts with rapid climb to peak flow followed by continuous decline of flows; (B) Failure to exert maximum efforts initially but can be considered valid; (C) Failure to exert maximally and test needs to be repeated; (D) Coughing during the first second of expiration and hence test should be repeated; (E) This shows knee and is a normal variant seen especially in young women; (F) Hesitation in the start and test should be repeated.

usually accompanies a strong forced exhalation effort. Hence, the vital capacity of the patient may well be much larger after an SVC test because there is little or no airway collapse during a controlled and slow exhalation effort. If the vital capacity improves after an SVC test, then it can be assumed that the original small FVC was caused by airway collapse and does not indicate the presence of restrictive disease. If the vital capacity does not improve either with the inhalation of a bronchodilator or does not improve with the administration of an SVC test, then restrictive pathologies must be considered as a possible cause for the small vital capacity results.

Q. What is the difference between FEV1 and FEF 25–75%?

Ans. FEV1 represents volume expired in 1 second, reflecting average flow over first second of expiration. FEF 25–75% is the average flow in the middle half portion of forceful

expiration, starting when 25% of FVC is exhaled and ending when 75% FVC is completed. Though both measurements are reduced in obstructive airway pathology, FEF 25–75% is supposed to reflect caliber of airways < 2 mm in diameter (small airway), as FEF 25–75% is measured at lower lung volumes. At lung volumes less than approximately 70% of FVC, flows become effort independent.

This effort independence is attributed to equal pressure point concept. During forced expiration, airway pressure gradually falls as air moves from distal most airways to proximal airway as airway caliber increases and corresponding resistance drops. At some point in the distal airway, intraluminal pressure falls below external pressure compressing airway (pleural pressure + lung recoil pressure) and the airway collapses trapping air. The greater the expiratory effort (pleural pressure), the more

proximal is equal pressure point. However, the occlusion of airway interrupts airflow; intraluminal pressure rises due to trapped gas and reopens the airway. Thus at equal pressure point, airflow is proportional to lung elastic recoil and inversely proportional to resistance of airway between alveoli and equal pressure point and not on pleural pressure.

Q. What are the advantages of flow-volume loop?

Ans.

- Flows at a particular point can easily be read from curve.
- Overall shape of curve can tell about adequacy of patient's effort and thus validity of report as mentioned prior.
- Small expiratory efforts with long concave shape indicate small airway obstruction.
- Large airway obstruction typically affects peak flow rates resulting in flat flow-volume loop and depending on site of obstruction flattening occurs either in both inspiratory and expiratory limb of loop or either limb.

Q. What is a diffusing capacity of the lung for carbon monoxide (DLCO)?

Ans. Diffusing capacity of the lung for carbon monoxide is a measurement of the ease of transfer for carbon monoxide molecules from alveolar gas to the hemoglobin (Hb) of the red blood cells in the pulmonary circulation. In Europe, it is often called the transfer factor of the lung for carbon monoxide (TLCO mmol/min/kilopascal), which describes the process more accurately. DLCO is a measure of the interaction of alveolar surface area, alveolar capillary perfusion, the physical properties of the alveolar capillary interface, capillary volume, Hb concentration, and the reaction rate of carbon monoxide and Hb. In principle, the total diffusing capacity of the whole lung is the sum of the diffusing capacity of the pulmonary membrane component and the capacity of the pulmonary capillary blood volume:

- *DLCO*: The capacity of the lungs to transfer carbon monoxide (mL/min/mm Hg)
- *DLCOc*: DLCO corrected for Hb (mL/min/mm Hg)
- *DLVA*: DLCO corrected for volume (mL/min/mm Hg/L)
- *DLVC*: DLCO corrected for both volume and Hb (mL/min/mm Hg/L).

Diffusing capacity of the lung for carbon monoxide may be decreased in anemic patient as Hb concentration is a very important measurement in interpreting reductions in DLCO. Because the level of Hb present in the blood and diffusing capacity are directly related, a correction for anemic patients (DLCOc) is used to further delineate whether a DLCO is decreased due to anemia or due to parenchymal or interface limitation:

- *Hemoglobin adjusted DLCO (adolescent males and men)*: $\text{Hb adjusted DLCO (DLCOc)} = \text{measured DLCO} [(10.22 + \text{Hb g/dL}) / (1.7 \text{ Hb})]$
- *Hemoglobin adjusted DLCO (children < 15 years and women)*: $\text{Hb adjusted DLCO (DLCOc)} = \text{measured DLCO} [(9.38 + \text{Hb g/dL}) / (1.7 \text{ Hb})]$.

Reduction in the diffusing capacity is classified as:

- *Mild*: Less than the lower limit of normal but > 60% of predicted
- *Moderate*: Between 40 and 60% of predicted
- *Severe*: <40%.

For risk stratification of postoperative pulmonary complications based on DLCO, refer chapter on "Pneumonectomy".

Q. How is DLCO performed?

Ans. The most commonly used and standardized technique is the single-breath technique. In this technique, a subject inhales a known volume of test gas that contains 10% helium, 0.3% carbon monoxide, 21% oxygen, and the remainder nitrogen. The patient inhales the test gas and holds breath for 10 seconds. The patient exhales to wash out a conservative overestimate of mechanical and anatomic dead space. Subsequently, an alveolar sample is collected. DLCO is calculated from the total volume of the lung, breath-holding time and the initial and final alveolar concentrations of carbon monoxide. The exhaled helium concentration is used to calculate a single-breath estimate of TLC and the initial alveolar concentration of carbon monoxide. Patient should avoid smoking for several hours before the test. Alcohol vapors can affect the accuracy of some fuel cell types of carbon monoxide analyzers; therefore, alcoholic beverages should be withheld for 8 hours prior to test.

Q. Are there any contraindications to perform DLCO?

Ans. Inability to follow instructions is a contraindication to a DLCO test. Patients should be alert, oriented, able to exhale completely and inhale to TLC, able to maintain an airtight seal on a mouthpiece and able to hold a large breath for 10 seconds.

Q. What are the conditions in which DLCO is affected?

Ans.

Decreases in DLCO

- *Obstructive lung diseases*: Emphysema, cystic fibrosis.
- *Parenchymal lung diseases*: Interstitial lung disease caused by fibrogenic dusts like asbestosis, biologic dusts like allergic alveolitis, drug reactions like amiodarone, bleomycin, idiopathic, sarcoidosis.
- *Pulmonary involvement in systemic diseases*: Systemic lupus erythematosus, progressive systemic sclerosis,

mixed connective tissue disease, rheumatoid arthritis, dermatomyositis-polymyositis, Wegener's granulomatosis.

- **Cardiovascular diseases:** Acute myocardial infarction, mitral stenosis, primary pulmonary hypertension, pulmonary edema, acute and recurrent pulmonary thromboembolism, fat embolization.
- **Other:** Diseases associated with anemia, chronic renal failure, chronic hemodialysis, marijuana smoking, cigarette smoking, bronchiolitis obliterans with organizing pneumonia (BOOP).

Increases in DLCO

Diseases associated with polycythemia, pulmonary hemorrhage, diseases associated with increased pulmonary blood flow such as left-to-right intracardiac shunts and exercise.

Q. What is an exercise-induced bronchoconstriction test?

Ans. Incidence of exercise-induced bronchoconstriction is observed in 70–80% of patients with asthma. Before performing this test, bronchodilators are withheld. Spirometry is then performed before and after 5, 15 and 30 minutes postexercise. A positive test is diagnosed when FEV₁ and FVC are reduced by 15% after exercise as compared to before exercise. Exercise protocol is like stress test on treadmill where 80% of maximum heart rate is targeted to terminate exercise.

Q. How would you diagnose site of obstruction from flow-volume loops?

Ans. *Fixed obstruction of upper airway (e.g., tracheal stenosis, bilateral vocal cord paralysis, goiter):* In fixed obstruction of the upper airway (**Fig. 4**), flow is limited by the caliber of the narrowed segment rather than by dynamic compression, resulting in equal reduction of inspiratory and expiratory flow rates. The top and bottom of loop are flattened so that the configuration approaches that of a rectangle. The fixed obstruction limits flow equally during inspiration and expiration and maximum expiratory flow (MEF) = maximum inspiratory flow (MIF).

Variable extrathoracic obstruction (e.g., single vocal cord paralysis, tracheomalacia or neoplasm invading trachea): When a single vocal cord is paralyzed, it moves passively in accordance with pressure gradients across the glottis. During a forced inspiration, it is drawn inward, resulting in decreased inspiratory flow. During a forced expiration, it is passively blown aside and expiratory flow is unimpaired, i.e., MIF 50% FVC is < MEF 50% FVC. During normal inspiration, airways within the thorax tend to dilate as the lung inflates while airways outside the thorax tend to

collapse due to negative intraluminal pressure. During expiration, the reverse is true as airways within the thorax collapse but airways outside the thorax are held open by expiratory flow. As a result, a variable extrathoracic obstruction leads to flattened inspiratory limb with normal expiratory portion (**Fig. 5**).

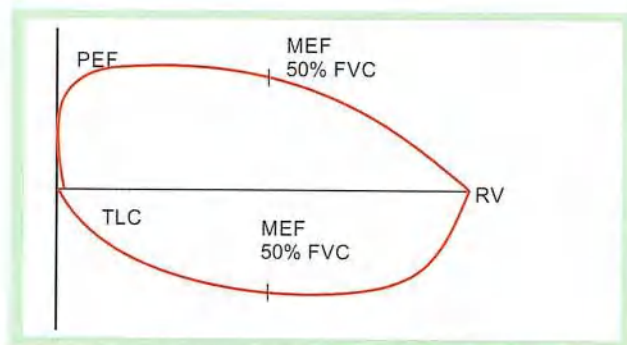


Fig. 4: Fixed obstruction of upper airway. (FVC: forced vital capacity; MEF: maximum expiratory flow; PEF: peak expiratory flow; RV: residual volume; TLC: total lung capacity).

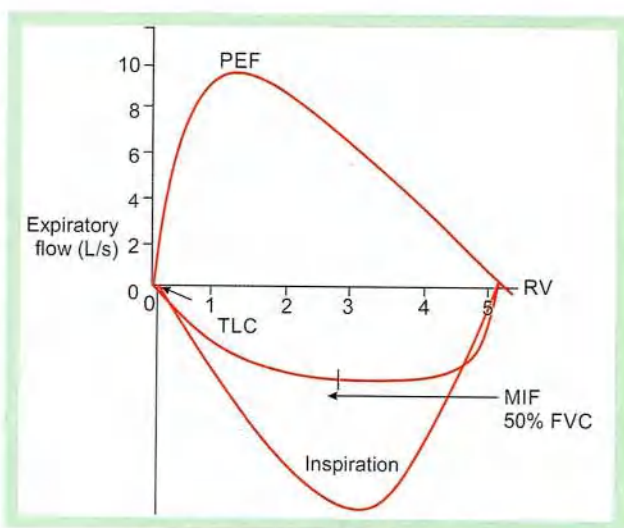


Fig. 5: Variable extrathoracic obstruction. (FVC: forced vital capacity; MIF: maximum inspiratory flow; PEF: peak expiratory flow; RV: residual volume; TLC: total lung capacity).

Variable intrathoracic obstruction (e.g., distal tracheomalacia, bronchomalacia): During a forced inspiration, negative pleural pressure holds the “floppy” trachea open. With forced expiration, the loss of structural support results in narrowing of the trachea and a plateau of diminished flow (a brief period of maintained flow is seen before airway compression occurs). Thus, a variable intrathoracic obstruction mainly affects the expiratory limb, again giving a flattened appearance to that aspect of the loop (**Fig. 6**).

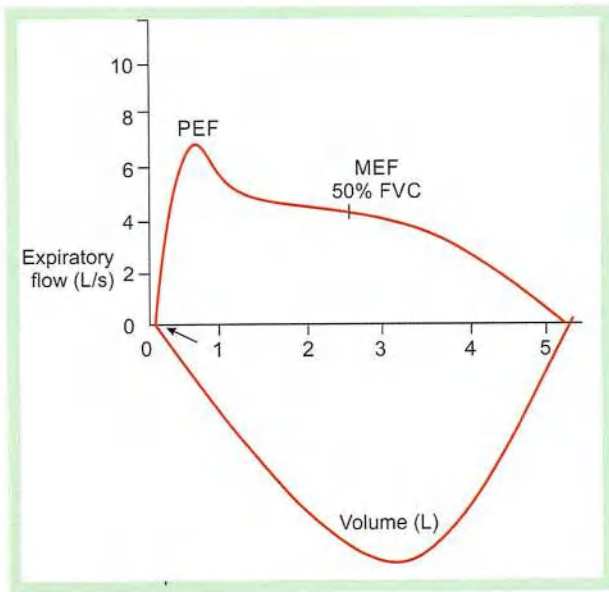


Fig. 6: Variable intrathoracic obstruction.

(FVC: forced vital capacity; MEF: maximum expiratory flow; PEF: peak expiratory flow).

CASE 1

A 67-year-old male, nonsmoker, presents with carcinoma stomach for total gastrectomy.

He has a past history of pulmonary tuberculosis for which he has taken complete treatment. His effort tolerance is one flight of stairs.

On clinical examination—he is pale, pulse rate (PR)—88 beats/min, regular blood pressure (BP): 140/86 mm Hg. His spirometry reveals:

Spirometry	Predicted	Actual	% predicted
FVC	3.65	2.43	67
FEV1	2.83	2.08	73
FEV1/FVC	75	86	114

Q. How would you interpret these values?

Ans. Forced vital capacity, FEV1 both are reduced, but FEV1/FVC is normal/increased. There is no obstructive element. This is suggestive of mild restrictive disorder.

Q. How will you optimize this patient? What care is needed postoperatively?

Ans. This is a mild disorder; probably due to his old healed Koch's which leads to fibrosis and typical restrictive disorder:

- Check room air saturation, ABG if required (very unlikely to be abnormal).
- Bronchodilators are not of much help in restrictive pattern but if the patient has simultaneous obstructive pattern then he should be started with it.
- *Chest physical therapy (PT)/deep breathing exercises (DBEs)*: This patient will have upper abdominal laparotomy where there is higher incidence of postoperative pulmonary complications. It is important that patient has good cough in postoperative period. Cough is related to IC/vital capacity, for which deep breathing would be of help. Epidural analgesia should be considered.
- Counsel the patient regarding the postoperative outcome possibility of postoperative ventilation (though remote as his PFTs are just mildly deranged).

Postoperative Care

- Maintain oxygen saturation (SpO_2) > 92%. Give supplemental O_2 with saline nebulization.
- Early mobilization.
- Chest PT/DBE.

CASE 2

A 60-year-old male presents with history of tobacco chewing and beedi smoking for 40 years. He is diagnosed with carcinoma of gallbladder and for radical cholecystectomy.

Spirometry	Predicted	Prebronchodilator actual	Prebronchodilator % predicted	Postbronchodilator actual	Postbronchodilator % predicted	Postbronchodilator % change
FVC	3.37	2.5	74	2.77	82	11
FEV1	2.69	1.33	49	1.89	70	42
FEV1/FVC	76	53	70	68	90	28
FEF 25–75%	3.24	0.58	18	1.47	45	153
MVV	118	42	35	57	48	37

Q. How would you interpret these values?

Ans. Forced vital capacity, FEV1, FEV1/FVC and FEF 25–75% are all reduced. MVV is decreased as well. This is suggestive

of an obstructive pattern. Also a significant reversibility is noted postbronchodilator administration.

Q. How will you optimize this patient?*Ans.*

- Start/continue bronchodilators and optimize the patient on bronchodilators
- Inhaled steroids
- Chest PT/DBEs.

Q. How will you manage this patient in the perioperative period?*Ans.*

- Smooth induction and maintain deep planes on anesthesia.
- Monitor peak airway pressures (if patient develops bronchoconstriction in intraoperative period, peak airway pressures will rise).
- Since this patient is a chronic smoker, he will have some elements of COPD also. Hence while ventilating this patient, keep lower respiratory rates and longer expiratory time to avoid auto-positive end-expiratory pressure (auto-PEEP).
- Intraoperative ABG would be of help to know gas exchange status.
- If patient develops bronchospasm during intraoperative period, one can use metered dose inhalers (MDIs). The circuit should not be disconnected and puffs given as the drug will not reach the lungs. For this purpose, a separate MDI adaptor should be attached to inspiratory limb of circuit before the "Y" piece. Puffs should be given through this adaptor synchronized with inspiration. The catheter mount acts as a spacer.
- One can use intravenous (IV) aminophylline keeping in mind its narrow safety margin and side effects like tachycardia, tachyarrhythmias.

Postoperative Care

- Continue inhaled/parental bronchodilators as per preoperative dose schedule.
- Supplemental O₂ to keep SpO₂ > 92%.
- Continue chest PT/DBEs/incentive spirometry.
- Venous thromboembolism prophylaxis.

■ CASE 3

A 49-year-old male patient a known case of sarcoidosis suffering from carcinoma tongue is planned for hemiglossectomy.

Spirometry	Predicted	Actual	% predicted
FVC	2.70	1.88	70
FEV1	2.30	1.72	75
FEV1/FVC	80	91	114
MVV	92	110	119
DLCUnc	20.25	11.05	55

*Contd...**Contd...*

Spirometry	Predicted	Actual	% predicted
DLCor	20.25	10.95	55
DL/VA	6.10	3.46	57
VA	4.61	3.17	69

Q. How would you interpret these values?

Ans. Forced expiratory volume in 1 second and FVC are both reduced but the FEV1/FVC ratio is increased suggestive of mild restrictive pattern.

The diffusing capacity is also lost suggestive of loss of functional alveolar capillary membrane.

It should be remembered that in patients with interstitial lung disease spirometry values can be misleading.

The diffusing capacity decreases much before the changes in the spirometry and thus this study should be carried on regular basis for patients suffering from interstitial lung diseases to know progress of disease.

Q. How would you optimize this patient?*Ans.*

- Baseline check SpO₂, ABG.
- Treatment for interstitial lung disease includes: corticosteroid drugs, cytotoxic drugs like azathioprine, antifibrotic drugs like penicillamine. As disease advances they require oxygen therapy. Patients with very severe disease require lung transplant.
- Continue all drugs in perioperative period.
- This patient will require higher fraction of inspired oxygen (FiO₂) in preoperative.
- Careful premedication only in monitored environment with O₂ supplementation.

Intraoperative Care

- Maintain SpO₂ and end-tidal carbon dioxide (EtCO₂) to the baseline level. Higher FiO₂ may be required.
- Maintain a high index of suspicion for concomitant pulmonary hypertension. Echocardiography would help diagnose right heart function.
- Strict attention to fluid balance. Avoid fluid overload.

Postoperative Care

- Maintain SpO₂ > 92%. Give supplemental O₂ if required.
- Progressive ventilatory compromise in these patients can lead to progressive hypoxia and hypercapnia which can be magnified by surgery and postoperative analgesia especially narcotics. Preoperative ABG should be done to know the baseline values and thus perioperative ventilatory strategies.
- Continue medications as per schedule.
- Chest PT/DBEs/incentive spirometry.
- Early mobilization.

- High dependency unit (HDU)/intensive care unit (ICU) care.

■ SUGGESTED READING

1. Ferguson MK, Dignam JJ, Siddique J, Vigneswaran WT, Celauro AD. Diffusing capacity predicts long-term survival after lung resection for cancer. *Eur J Cardiothorac Surg*. 2012;41:e81-6.
2. Gildea TR, McCarthy K. (2018). Disease management project: Pulmonary function testing. [online] Available from: <http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/pulmonary/pulmonary-function-testing> [Last accessed February, 2020].
3. Kaminsky DA, Whitman T, Callas PW. DLCO versus DLCO/VA as predictors of pulmonary gas exchange. *Respir Med*. 2007;101:989-94.
4. Kreider ME, Grippi MA. Impact of the new ATS/ERS pulmonary function test interpretation guidelines. *Respir Med*. 2007;101:2336-42.
5. McCarthy K, Dweik RA. (2018). Pulmonary function testing. [online] Available from: <http://emedicine.medscape.com/article/303239-overview#aw2aab6b4> [Last accessed February, 2020].

Anesthesia Machine

Nayana Amin, Vandana Agarwal

Q. Describe the parts of anesthesia machine.

Ans. Anesthesia machine controls the release of pressurized gases at preset rates, adds vapor to the gases as required and delivers the mixture to a common gas outlet. Pressurized gases are supplied by cylinders or pipelines to the anesthetic machine. Continuous flow anesthetic machines date back to the first availability of compressed gases, and despite numerous modifications the modern apparatus retains many of the features of the original Boyle's machine, a British Oxygen Company trade name in honor of the British anesthetist HEG Boyle (1875–1941).

The basic structure of anesthesia machine has a steel framework which is mounted on four wheels. The machine can be divided into three parts:

High pressure system consists of all parts of the machine which receive gases at cylinder pressure. It includes:

- The hanger yoke which connects the cylinder to the machine, yoke block which is used to connect cylinders larger than size E or pipeline hoses to the machine through the yoke.
- The cylinder pressure gauge.
- The pressure regulator which converts the high variable gas pressure into a lower more constant pressure, suitable for use in the machine.

The intermediate pressure system includes components of the machine which receive gases at reduced pressures. It begins at the pipeline inlet pressures of 60 psig (50–55 psig in the US) and regulated cylinder pressures of 45 psig to the flow control valves where the pressures would be 14–26 psig.

The parts of the intermediate pressure system are as follows:

- Pipeline inlet connections which connect the machine to the hospital pipeline system.
- Pipeline pressure gauges.
- Ventilator power outlets which supplies driving gas to the ventilator.

- Oxygen pressure failure devices which either interrupt the flow of anesthetic gases or provide an alarm when oxygen supply fails.
- Second stage pressure regulators.
- Oxygen flush which allows delivery of high flows of oxygen from the machine.
- Flow control valves.
- Auxiliary oxygen flowmeters.

Low pressure system takes the gases from the flowmeters to the machine outlet. It is the part of the machine downstream of the flowmeters in which the pressure is slightly above atmospheric pressure. The components of the low pressure system are as follows:

- Flowmeters
- Antihypoxia devices
- Vaporizers
- Vaporizer circuit control valves
- Back pressure safety devices
- Outlet check valve
- Common gas outlet.

Q. What are the various units for expressing pressure? What are their conversion factors?

Ans. The various units of pressure and their conversion factors are as follows:

$$100 \text{ kPa} = 1000 \text{ mbar} = 760 \text{ mm Hg} = 1030 \text{ cm H}_2\text{O} = 14.7 \text{ psi} = 1 \text{ atmosphere}$$

Therefore,

$$1 \text{ kPa} = 10 \text{ mbar} = 7.6 \text{ mm Hg} = 10.3 \text{ cm H}_2\text{O} = 0.147 \text{ psi}$$

Q. How do you define compressed gas?

Ans. A compressed gas is defined as any mixture having in a container an absolute pressure exceeding 40 psi at 70°F, or regardless of the pressure at 70°F, having an absolute pressure exceeding 104 psi at 130°F, or any liquid having a vapor pressure exceeding 40 psia at 100°F.

Q. Which are the compressed gases used in anesthesia?

Ans. Both liquefied and nonliquefied compressed gases are used in anesthesia. The nonliquefied compressed gases are oxygen, nitrogen, air, and helium. These gases do not liquefy at ordinary ambient temperatures no matter how much pressure is applied. Liquefied compressed gases are gases which become liquid at ambient temperatures and at pressures ranging from 25 to 2500 psig. Examples are nitrous oxide and carbon dioxide.

Q. What do Psi, Psig, and Psia stand for?

Ans. Psi stands for pounds per square inch.

Psig stands for pounds per square inch gauge. It is the difference between the measured pressure and surrounding atmospheric pressure.

Psia stands for pounds per square inch absolute. Zero pressure for a perfect vacuum is taken as a reference point. Therefore, $Psia = psig + \text{local atmospheric pressure}$.

Q. What is the difference between gas and vapor?

Ans. A gas is a substance which is in the gaseous state at room temperature and atmospheric pressure as its critical temperature is below the room temperature.

A vapor is a gaseous substance which is below its critical temperature under ambient conditions. It exists as a liquid at room temperature and atmospheric pressure as its critical temperature is above the room temperature.

Q. What do you mean by critical temperature? What is the critical temperature of oxygen and nitrous oxide?

Ans. Critical temperature is the temperature above which any gas cannot be liquefied no matter how much pressure is applied. The critical temperature of oxygen is -119°C due to which it exists as a gas in a cylinder at room temperature. The critical temperature of nitrous oxide is 36.5°C due to which it exists as a mixture of liquid and gas in a cylinder at room temperature. If the ambient temperature exceeds 36.5°C then nitrous oxide can only exist in the gaseous state.

Q. What is critical pressure?

Ans. Critical pressure is the minimum pressure that is required to liquefy a gas at its critical temperature.

Q. How and why is bulk oxygen storage done?

Ans. If oxygen consumption is very high then it is more economical to store oxygen in liquid form as a liquid would occupy only 1/860 of the space the gas would occupy. At a temperature of 15°C and atmospheric pressure, largest size J cylinder will give approximately 130 times its capacity of oxygen while one volume of liquid oxygen will give approximately 840 times its capacity of oxygen. The liquid oxygen is stored in special designed vessel called vacuum insulated evaporator (VIE). It has an outer shell made of carbon steel and an inner shell made of stainless steel. The two shells are separated by vacuum so that there is minimal temperature fluctuation in the inner shell. Oxygen is in a liquid form because the temperature inside the chamber is -153°C which is lower than its critical temperature. If there is an increase in temperature it will result in evaporation of the liquid oxygen. If oxygen is not being used it can result in pressure build up in the chamber, which is prevented by venting the excess gas through a safety valve. The pressure in an oxygen cylinder is 1,900 psi whereas the pressure in a liquid oxygen tank is only 20–90 psi. There is gaseous oxygen above the liquid oxygen. When oxygen is being used it is first heated and the pressure is regulated to the pipeline pressure before delivering it to the hospital pipeline system.

Q. What are cylinders made of?

Ans. Older cylinders were made of carbon steel and were heavier. Modern cylinders are made from alloy containing molybdenum and steel. As molybdenum steel is stronger the cylinders have thinner walls and therefore are lighter. MRI compatible cylinders are made of aluminum. The wall thickness is 5/64 to 1/4 of an inch. In India, the cylinders are made of manganese steel.

Q. What are the different sizes of cylinders and what are their capacities?

Ans. Cylinders are available in various sizes. They are designated according to alphabets (size A to J). Size A is the smallest size. Size E is most commonly used in the anesthesia machine. The pressures and capacities of different sizes of medical gas cylinders are mentioned in **Table 1**.

TABLE 1: Sizes of cylinders and their content.

		Oxygen	Nitrous oxide	Air	Nitrogen	Carbon dioxide
Size D	Content in L	400	940	375	370	940
	Pressure in psig (kPa)	1,900 (13,700)	745 (4,400)	1,900 (13,700)	1,900 (13,700)	838 (5,000)
Size E	Content in L	660	1,590	625	610	1,590
	Pressure in psig (kPa)	1,900 (13,700)	745 (4,400)	1,900 (13,700)	1,900 (13,700)	838 (5,000)
Size H	Content in L	6,900	15,800	6,550	6,400	
	Pressure in psig (kPa)	2,200	745 (4,400)	2,200	2,200	

Q. What do you mean by tare weight? What is the tare weight of size E oxygen and nitrous oxide cylinder?

Ans. Tare weight is weight of an empty cylinder. Tare weight of nitrous oxide cylinder is 7 kg.

Q. What is the internal volume of cylinders of various sizes?

Ans. Sizes: Size D- 3 L, Size E- 5 L, Size F- 10 L, Size H- 46-50 L.

Q. How can you measure the contents of a cylinder?

Ans. In a cylinder containing nonliquefied gas such as oxygen, the pressure in the cylinder decreases as the contents are used. Therefore the pressure on the pressure gauge can be used to measure the cylinder contents. The volume of the contents available at atmospheric pressure can be estimated using Boyle's law which states that at constant temperature pressure is inversely proportional to volume. Therefore if the temperature is constant then according to Boyle's law $P_1 \times V_1 = P_2 \times V_2$.

Example: We know that the volume of size E cylinder is 5 L (V_1). If the pressure gauge reads 1,000 psi (P_1) then to know how much of oxygen (V_2) will evolve at atmospheric pressure of 14.7 psia (P_2) we have to apply Boyle's law.

$1,000 \times 5 = 14.7 \times V_2$, therefore, $V_2 = 1000 \times 5 \div 14.7 = 340$ L.

If pressure gauge reads 600 psi then $V_2 = 600 \times 5 \div 14.7 = 204$ L.

Another Method

A full cylinder at 1,900 psi will evolve 660 L of oxygen, then at a pressure of 1,000 psi how much oxygen will evolve? $1,000 \div 1,900 = 52\%$ full, hence it will evolve $660 \times 52\% = 340$ L.

If pressure gauge reads 600 psi then it is 31% full and it will evolve $660 \times 31\% = 204$ L.

If the cylinder contains a liquefied gas such as nitrous oxide then the pressure in the cylinder would not be indicative of the contents of the cylinder. This is because the pressure in the cylinder depends on the vapor pressure of the liquid gas. As a result, the pressure would remain constant until all the liquid has evaporated, after which the pressure decreases as the cylinder empties. Hence, for liquefied gases the weight of the contents of the cylinder can also be estimated by weighing the cylinder and subtracting the weight of the empty cylinder or the tare weight.

By Avogadro's law, 1 g molecular weight (i.e., the molecular weight in grams) of any gas or vapor occupies 22.4 L at standard temperature and pressure [STP: 0°C (which is 273° absolute or 273 K) and 760 mm Hg pressure (or one atmosphere)]. According to Charles' Law, at a constant pressure the volume of a fixed mass

of gas is directly proportional to absolute temperature. Room temperature is usually 20°C or 293 K (i.e., $273 + 20$). Therefore, 32 g of oxygen or 44 g of nitrous oxide will occupy $22.4 \times 293 \div 273$ or 24 L at 20°C .

Example: How much nitrous oxide will evolve from a cylinder weighing 8 kg?

Assume tare weight of size E nitrous oxide cylinder is 7 kg. The pressure gauge reads 745 psi and the weight of the cylinder is 8 kg then how much nitrous oxide will evolve at 20°C at atmospheric pressure. The mass of nitrous oxide is 1,000 g (8 kg-7 kg). We already know that 44 g of nitrous oxide will occupy 24 L.

The amount of gas that will evolve from 1,000 g will be $1,000 \times 24 \div 44 = 545$ L.

If the weight of the cylinder is 7.5 kg then the mass of nitrous oxide is 500 g and the amount of gas evolved will be $500 \times 24 \div 44 = 272$ L.

If all the liquid nitrous oxide has been used up and the cylinder contains only gaseous nitrous oxide then at this point we can apply Boyle's law to know how much nitrous oxide will evolve at atmospheric pressure. If cylinder contains only nitrous oxide gas and has a pressure of 745 psi (P_1).

Then, $745 \times 5 = 14.7 \times V_2$, therefore $V_2 = 745 \times 5 \div 14.7 = 253$ L. When the cylinder contains only nitrous oxide gas, it is 16% full ($253/1,590$ L).

If the pressure gauge reads 300 psi then it will evolve $300 \times 253 \div 745 = 100$ L.

Q. Is the pressure gauge a reliable indicator of the contents of a cylinder?

Ans. The pressure gauge would be a reliable indicator of the contents of a cylinder containing nonliquefied compressed gases. However, it is not a reliable indicator of the contents of cylinders containing liquefied gases such as nitrous oxide. This is because while using a nitrous oxide cylinder the pressure can remain constant if the temperature is constant. A constant temperature would ensure the vapor pressure remains constant and the pressure would not change as the cylinder is being used. However, in practice the temperature does not remain constant. Heat energy is required for the evaporation of the liquid in the cylinder. As the temperature falls the vapor pressure also falls. There is a progressive fall in the cylinder pressure as the nitrous oxide is used. The rate at which the pressure falls is related to the flowrate.

Q. Why do you get frost on the outer surface of nitrous oxide cylinders?

Ans. When a liquefied gas such as nitrous oxide is being used, the latent heat of vaporization causes cooling of the cylinder. The fall in the temperature of the cylinder is

much more if the high flow rates of nitrous oxide are used. As the cylinder cools, water vapor from the surrounding can condense on the external surface of the cylinder. If the temperature is very low, the water vapor may even freeze resulting in frost formation on the outer surface of the cylinder. Cooling of nitrous oxide cylinder during use indicates that there is residual liquid nitrous oxide in the cylinder.

Q. How are the cylinders tested?

Ans. Periodic visual inspection of the cylinder to check for any distortion, cracks, dents, leaks, etc:

- *Tensile test* on 1 out of every 100 cylinders manufactured. Strips of the cylinder are cut and stretched.
- *Flattening test, bend test, and impact test* are carried out on at least 1 in every 100 cylinders.
- *Hydraulic test*: The cylinder is tested to 1.66 times its service pressure. This helps detect any leaks and to determine the retention of structural strength. A cylinder must be subjected to internal hydrostatic pressure testing at least every 5 years. With a special permit it can be extended up to every 10 years.

Q. What precautions should be taken while transporting a cylinder?

Ans.

- Upright position
- Wear protective footwear/safety gloves
- Do not lift by protective cap/guard as you may damage valve
- Ensure all cylinders are properly labeled as to the contents
- Do not subject cylinders to temperature extremes
- Do not drag slide or roll—use cylinder cart
- Cylinders should be secured at all times.

Q. What should you check while receiving a gas cylinder?

Ans. When a gas cylinder is received, it shall be checked for the following:

- A stamped hydraulic test date within the last 5 years
- A stenciled or labeled identification of its contents
- Cylinder is in an acceptable condition
- Presence of a valve protection cap.

Cylinders should be rejected if the test date, identification, markings or cap are not in order or if the cap is rusted or inoperable.

Q. What precautions should be taken while storing compressed gas cylinders?

Ans.

- Cylinders should not be stored in the operation theaters. There should be a designated area for storing cylinders.

- Storage area should be well-ventilated, cool and clean and made of fire-resistant materials.
- Larger cylinders should be stored in upright position and secured with chains. Smaller cylinders should be stored horizontally in racks.
- Cap cylinders when not in use.
- Ensure all cylinders are properly labeled as to the contents.
- Always secure cylinders with chain or cart.
- Do not secure more than four cylinders in any one row.
- Keep cylinders away from radiators and other sources of heat.
- Do not subject cylinders to temperature extremes.
- Separate storage of flammable and oxidizing cylinders by at least 20 feet or a fire-resistant barrier of at least five feet high and having a fire rating of at least 1 hour.
- Mark used cylinders "Empty". They should never be stored with other full cylinders.
- Do not store cylinders where water is freestanding or may collect.
- Do not store cylinders where they can become part of an electrical circuit.
- Do not store cylinders in hallways, closets or vestibules.
- Do not store cylinder in front of eyewash/emergency shower stations, spill kits or fire extinguishers.
- Cylinders should not be exposed to continuous dampness, corrosive chemicals or fumes.
- Cylinders should not be covered during storage. Wrappings should be removed from the cylinders during storage.

Q. What is the service pressure of a cylinder?

Ans. The service pressure is the maximum pressure to which the cylinder may be filled at 70°F.

Q. What are the filling limits for any cylinder? Why do you need filling limits?

Ans. When a gas is kept in a closed container, a rise in temperature will cause a rise in pressure. If a cylinder which has been filled to a safe pressure at normal temperature is subjected to very high temperatures, the pressure can increase to very dangerous levels. This can even cause the cylinder to explode. In order to prevent this from happening the Department of Transportation has established regulations limiting the amount of gas a cylinder may contain.

Filling Limits for Nonliquefied Gases

The pressure in the filled cylinder at 70°F should not exceed the service pressure marked on the cylinder. However,

some nonliquefied gases such as oxygen, helium, carbon dioxide-oxygen mixtures, helium-oxygen mixtures which may be allowed an additional 10%.

At 130°F, the pressure in cylinders containing non-liquefied gases may not exceed $1\frac{1}{4}$ times the maximum permitted filling pressures at 70°F.

Filling Limits for Liquefied Gases

In cylinders containing liquefied gases, the pressure will remain nearly constant as long as there is liquid in the cylinder. To prevent the cylinder containing liquefied gas from being overfilled, the maximum amount of gas that can be contained is defined by a filling ratio for each gas:

- Nitrous oxide—68%
- Carbon dioxide—68%
- Cyclopropane—55%.

Q. Define filling density (filling ratio).

Ans. Filling ratio is the percent ratio of the weight of the gas in the cylinder to the weight of the water a cylinder can hold at 60°F.

Q. Is the filling ratio for nitrous oxide same in all the countries?

Ans. In tropical countries, the filling ratio is 0.68 and in temperate climates it is 0.75.

Q. How would you identify the contents of a cylinder?

Ans. The cylinder contents can be identified by the color of the cylinder (**Table 2**) and the label on the cylinder. Each cylinder must have a conspicuous label which contains all the important information. It mentions the name, chemical symbol and physical state of the contents of the cylinder. It also mentions the maximum cylinder pressure, the cylinder contents in L, cylinder size code, and product licence number. The label not only helps to identify the contents of the cylinder but also gives a warning of the principal hazards associated with the cylinder or its contents. As there are many medical gases available, the cylinders are color coded for a specific gas as an added safety feature. The chemical symbol of the gas contained in the cylinder is marked on the cylinder valve.

TABLE 2: Identification of cylinders through color coding.

Gas	International	United States
Oxygen	Black body and white shoulders	Green
Nitrous oxide	Blue	Blue
Carbon dioxide	Gray	Gray
Air	Black body and shoulders with black and white quarters	Yellow
Helium	Brown	Brown

Contd...

Contd...

Entonox	Blue body and shoulders with blue and white quarters	
Cyclopropane	Orange	Orange

Just like United States there are many other countries which do not follow the international codes, hence, color coding is only an aid to identifying the cylinder contents and cannot be completely relied upon. The label is the primary means of identification. In India, we follow international color code.

Q. Can you describe the cylinder valve?

Ans. Cylinders are filled and discharged through the cylinder valves. The valves are attached to the neck of the cylinder by means of tapered threads. The valves are made of brass with chromium plating. Small cylinder valves are used for cylinder sizes A to E and large cylinder valves are used on cylinder sizes F to H. Each valve has the following parts:

- The body which forms the basic structure.
- The port is the point of exit for the cylinder contents. It fits over the nipple of the yoke (on small cylinders) or the regulator (on large cylinders). Any damage to the port may prevent a tight seal and result in a leak. Hence, the port should be covered during transit. While installing the cylinder if the port is mistaken for the conical depression on the opposite side of the valve then, inserting the retaining screw into the port may damage the port and the index pins.
- The stem or shaft, when rotated will either open or close the valve. The stem has to be rotated in the anticlockwise direction to open the valve and in the clockwise direction to close it.
- There is a handle or hand-wheel for turning the valve stem.
- Safety relief device, which allows discharge of cylinder contents to the atmosphere under certain conditions of exposure.
- There is a conical depression on the small cylinder valves just above the safety relief device. It receives the retaining screw of the yoke. If the retaining screw is tightened into the safety relief device then the device may be damaged and the cylinder contents may escape.
- The pin index is a noninterchangeable safety system which prevents the attachment of the incorrect cylinder to the yoke.

Q. What are the differences between direct and indirect acting valves?

Ans. Differences between direct and indirect acting valves are described in **Table 3**.

Most cylinder valves are of the packed type.

TABLE 3: Differences between direct and indirect acting valves.

Direct acting	Indirect acting
Packed valves	Diaphragm valves
Turning the stem causes the seat to turn	Turning the stem moves a metal diaphragm against the opposing force of spring acting on the seat assembly
Stem is sealed by resilient packing like Teflon	No packing
Less expensive	More expensive
Good performance with gases under high pressure	It is used for gases such as nitrous oxide which liquefy under pressure. These valves operate better at low temperatures which are produced when these liquids vaporize. Less wear and tear on the seat These valves are tighter at the stem and the seat does not turn, therefore, they are less susceptible to leaks
These valves open fully in 2–3 full turns	These valves open fully in $\frac{1}{2}$ to $\frac{3}{4}$ turns

Q. What safety feature in a cylinder valve protects against buildup of high pressure in the cylinder? Can you describe any one of them?

Ans. All cylinder valves have a safety relief device which is designed to prevent rupture of the cylinder under certain conditions of exposure. The safety relief devices are of three types namely frangible disk assembly, fusible plug and safety relief valve.

Frangible disk assembly: It consists of a frangible disk and a safety cap. The ports of this assembly may be filled with a fusible alloy. The disk is made of copper. This disk closes the discharge channel and prevents the release of the contained gas. The bursting pressure is the minimum pressure for which the disk is designed to burst so that the gas can escape through the discharge channel. The burst pressure is determined by the material, thickness and shape of the disk, and the diameter of the discharge channel. It is a nonreclosing device and is used in oxygen, air, nitrous oxide, carbon dioxide, nitrogen, helium, and carbon dioxide-oxygen and helium-oxygen cylinders. It protects against high pressures resulting from high temperature or overfilling.

Fusible plug: It is usually made of a metal alloy with low melting point. This alloy occludes the discharge channel and melts at a predetermined temperature. Wood's metal alloy is most commonly used. The yield temperature is the temperature at which the alloy will melt. It protects against excessive pressure resulting from high temperature but not from overfilling.

A combination of frangible disk and a low melting point fusible plug is available. This prevents bursting at a predetermined pressure unless the melting temperature has also been attained. This device does not protect against high pressure due to overfilling.

Safety relief valve: It consists of a valve seat which occludes the discharge channel. It is held in place by a spring. When the set pressure is exceeded the valve opens and the gas escapes through the vents in the safety valve. Once the pressure is reduced the valve closes again. This type of pressure relief device is more susceptible to leaks.

Q. What is wood metal?

Ans. Woods metal is a fusible alloy which contains bismuth, lead, tin, and cadmium. Depending on the composition it melts at two different temperatures either 70°C–75°C or 95°C–105°C. It is named after American metallurgist B Wood.

Q. Are there any other safety features on the cylinder valve?

Ans. The pin index safety system on the cylinder valve prevents an incorrect cylinder from being attached to the anesthesia machine. It was first used in 1952. This is an additional safety feature as the color coding of cylinders is not a foolproof method of protecting against human error.

Q. What is the pin index safety system?

Ans. The pin index safety system is used in small cylinder valves (size E or less) with yoke type connections. It consists of two pins projecting from the inner surface of the yoke. They are positioned in such a way that they fit into two corresponding holes drilled into the cylinder valve. The holes are positioned in an arc below the outlet port on the cylinder valve. The arc has a radius of 9/16 inch. The six holes are placed at an angle of 12° from the port, while the seventh hole is at the middle of these holes. The port will not seat against the washer of the yoke unless the pins and holes are aligned. The pins are 4 mm in diameter and 6 mm long. Pin 7 is slightly thicker (**Fig. 1**).

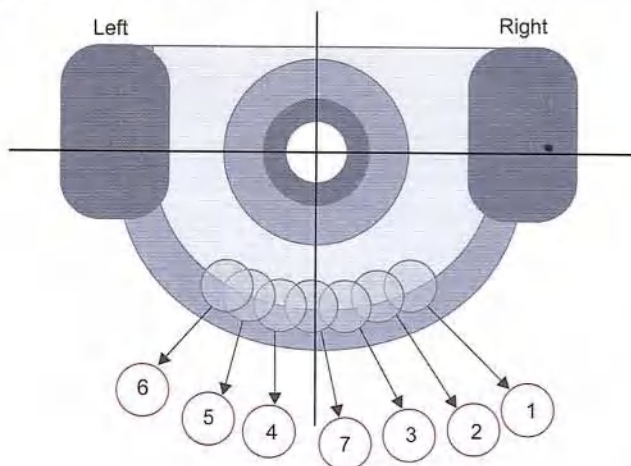


Fig. 1: Pin index safety system.

The pin index for different gases is as follows (Table 4):

Gas	Pin index
Oxygen	2.5
Nitrous oxide	3.5
Carbon dioxide	1.6
Air	1.5
Cyclopropane	3.6
O ₂ -CO ₂ (CO ₂ < 7.5%)	2.6
O ₂ -CO ₂ (CO ₂ > 7.5%)	1.6
O ₂ -He (He > 80.5%)	4.6
O ₂ -He (He < 80.5%)	2.4
Entonox	7

The pin index system is also used when the cylinders are being refilled. This prevents a wrong gas from being filled into the cylinder.

Q. What are the fallacies of the pin index safety system?

Ans. In spite of having a pin index system, one can still attach a wrong cylinder if the pins on the yoke are broken. If someone places two washers on the port, then the pin index would be nullified and a wrong cylinder can be placed on the yoke.

Q. What is the safety feature for valve outlet connections for large cylinders?

Ans. As the large cylinders cannot directly connect to the yoke, the pin index system cannot be used. The valves of large cylinders have threaded outlet which mesh with those of the nut. When the nut is turned clockwise the nipple seats against the valve outlet and the gas channel of the valve is aligned with the channel of the nipple. Different gas cylinders will have different threaded valves. The valve threads may be external, internal, right hand or left hand.

Also the diameters of the threads vary. Further separation is made by varying the size and shape of seats and nipples for any given thread size.

Q. Why do you need to open the cylinder valve slowly?

Ans. When the cylinder valve is opened very fast, the gas quickly passes into the space between the cylinder valve and the yoke or regulator. The sudden recompression of the gas generates large amounts of heat (Joule-Thomson effect). Since there is very little time for the dissipation of heat, it results in an adiabatic process (heat is neither gained nor lost from the environment). If there is any dust or grease in this space, it can be ignited by the heat causing flash fire or explosion. When the valve is opened slowly, the recompression of the gas is prolonged. This allows some time for the dissipation of heat thereby reducing the risk of fires and explosions. Also when the valve is opened rapidly the sudden release of gases may damage the pressure gauge and regulator attached to the machine. Before the cylinder is fitted to the machine the valve should be opened slowly till the pressure on the gauge stabilizes. The gas which escapes helps to blow off the dirt present on the valve outlet. This dirt can damage the pressure regulator or even cause an explosion.

Q. If operation theater attendant gives a suggestion of applying grease on the cylinder valve which is too tight what will you do?

Ans. You should not apply grease on the valve cylinder the reasons have already been mentioned above.

Q. What do you mean by cracking the valve?

Ans. Before connecting the cylinder to the anesthesia machine the protective cap should be removed and the valve should be opened slowly and briefly. While opening the valve the port should point away from the user or other personnel. This is known as cracking the valve. It helps to clear dust and other foreign materials from the valve outlet before it is connected to the machine. It reduces the possibility of fires and explosions when the valve is opened after connecting it to the machine. It also prevents the dust from being blown into the machine and clogging the filters.

Q. Is it a safe practice to keep cylinder valve open at all times?

Ans. No, the cylinder valve should not be kept open at all times as it can result in slow depletion of gases from the cylinder. Usually the pipeline pressures are higher than the regulated cylinder pressures. If the cylinders are kept open, then whenever the pipeline pressure drops below the cylinder pressure (during the use of ventilators or oxygen flush) gases will preferentially be used from the cylinder. This will result in depletion of gases from the cylinder and no

reserve will be available if the pipeline supply fails. Hence the cylinder should only be opened while checking the cylinder or when the pipeline is not available.

Q. Can you summarize the safety features of cylinders?

Ans.

- Label on the cylinder
- Pin index safety system
- Safety relief valve on the cylinder
- Symbol of the gas on the cylinder valve
- Color coding.

Q. What is Entonox?

Ans. Entonox is the trade name for a compressed gas mixture containing 50% oxygen and 50% nitrous oxide by volume. Entonox is stored in cylinders at a pressure of 13,700 kPa (1,900 psig). It is mainly used for labor analgesia.

Q. What is the Poynting effect?

Ans. Poynting effect or overpressure effect is the change in the critical temperature and pressure of one gas when it is mixed with another gas. Poynting effect is seen in Entonox cylinders which contains a mixture of 50:50 of nitrous oxide and oxygen. Normally oxygen is stored in the cylinder in a gaseous state as its critical temperature is very low (-119°C) whereas nitrous oxide is stored as a liquid. When gaseous oxygen is bubbled through liquid nitrous oxide there is vaporization of the liquid resulting in the formation of mixture of oxygen and nitrous oxide in a gaseous state. Thus due to the Poynting effect nitrous oxide which is normally present in cylinders as liquid, is present as a gas in the mixture with oxygen. The Poynting effect reduces the critical temperature of N_2O so Entonox has a pseudocritical temperature of -6°C . The physical property of Entonox gas is different from the individual gases.

Q. What do you mean by lamination and what are its clinical implications?

Ans. The physical property of Entonox gas is different from the individual gases. Entonox exists in a gaseous state and the pressure in a full Entonox cylinder is 1,900 psig (13,700 kPa). The Entonox cylinders should be stored in horizontal position at a temperature between 10°C and 38°C . It is important to store Entonox cylinders above its pseudocritical temperature of -6°C . If the temperature drops below -6°C there is liquefaction and separation of its two components. This is known as lamination. There is a liquid mixture which mostly contains nitrous oxide with about 20% oxygen dissolved in it. Above this liquid is a gas mixture containing a high concentration of oxygen. As the cylinder is being used initially a high concentration of oxygen will be supplied. As the liquid evaporates the concentration of oxygen will go on decreasing. When

the cylinder is nearly empty the patient will get hypoxic mixtures containing $<20\%$ oxygen. This process can be reversed by rewarming the cylinder and mixing its contents. If a cylinder has been exposed to cold below -6°C it should be warmed for 5 minutes in a 37°C water bath or for 2 hours in a room at 15°C . It should then be inverted three times before use.

Q. How can liquefaction and separation of Entonox be prevented?

Ans. To prevent liquefaction and separation of Entonox the cylinders should be stored in horizontal position at a temperature of 5°C for at least 24 hours before use. The horizontal position increases the area of diffusion. The cylinder should also be inverted several times before use. In large cylinders there is a dip tube with its tip ending in the liquid phase. This ensures that the liquid is used first, thus preventing the delivery of oxygen at concentration $<20\%$.

Q. Can you describe the hanger yoke assembly?

Ans. The hanger yoke orients and supports the cylinder, provides a gas tight seal and ensures unidirectional flow of gases to the machine. It consists of the following parts:

- *The body* is the main framework and supporting structure. It has a toggle handle or swivel gate which cannot be closed if the cylinder valve is not properly placed.
- *The retaining screw* tightens the cylinder into the yoke and helps to establish a seal. The tip of the retaining screw fits into the conical depression on the cylinder valve.
- *The nipple* is the part of the yoke through which the gas enters the machine. It fits into the port on the cylinder valve.
- *The index pins* prevent the attachment of an incorrect cylinder to the yoke.
- *The washer* helps to form a seal between the cylinder and the yoke.
- *Filter* is present between the cylinder and the regulator to prevent particulate matter from entering the machine and damaging the components of the machine.
- *Check valve assembly*, which ensures a unidirectional flow of gases through the yoke. It prevents the flow of gases from the machine or from another cylinder to atmosphere when the yoke is empty. When double yokes are present the check valves prevent transfer of gas from a cylinder at high pressure to a cylinder at low pressure. It also allows an empty cylinder to be exchanged with minimal loss of gas, even as the gas flow continues from the other cylinder.

Q. What are the fallacies of the check valve assembly?

Ans. The check valves do not act as permanent seals for empty yokes. Small amounts of gas can escape especially

if the cylinder pressure is low. In order to minimize the losses, the yokes should not be left vacant for extended periods. An empty cylinder should be replaced as soon as possible. The valve of the empty cylinder should be closed if it cannot be replaced. A yoke plug can also be used to prevent gas leak. Also if the check valve is defective then transfilling of the cylinders can occur. In order to avoid this only one cylinder should be opened at a time.

Q. Can you enumerate the uses of yoke block?

Ans. Yoke blocks are a piece of metal-shaped like a cylinder valve. They have a port and conical depression to fit the yoke. They are also pin indexed. They are used to:

- Connect cylinders larger than size E to the machine
- To connect pipeline supply to the machines that does not have pipeline connections
- They are used to block the yoke when there are no cylinders present. This helps prevent small gas leaks from the machine if the check valves are defective.

Q. Why are yoke blocks no longer used nowadays?

Ans. The use of yoke blocks has been associated with several hazards and hence is no longer used nowadays. If the yoke block is used to connect a high pressure cylinder without a regulator, then the hose carrying the gas at high pressure can pose a hazard to personnel if it leaks or breaks. It is safer to use pipeline inlet connections than yoke blocks because yoke blocks can be responsible for crossover of gases in the anesthesia machine. Some yoke blocks do not have pin index system and can be inserted into any yoke. The pin indexed yoke can be inserted upside down if they have a short top. The pressure regulators in the machine which are designed to accept gases at cylinder pressure may not function properly when supplied with gases at reduced pressure. This can lead to fluctuations of gas pressure supplied to the flow control valve. Due to these reasons yoke blocks are no longer used and pipeline inlet connections are used whenever cylinders larger than size D or E are used.

Q. Can you describe the cylinder pressure gauge?

Ans. The cylinder pressure gauge is Bourdon tube type. It indicates the pressure in the cylinder when the cylinder valve is opened. It has a hollow metal tube which is bent into a curve. One end is sealed and the other end is open. The sealed end is connected to a clock-like mechanism. The open end is connected to a gas source and soldered into a socket. There is a constriction at the entry point of the gas. This helps to prevent any pressure surges that can damage the gauge. It also prevents rapid loss of the gas when the pressure gauge ruptures. When the cylinder valve is opened the gas enters the tube. The high pressure in the tube causes it to straighten. The movement of the sealed end is transmitted to the indicator attached to the clockwork

mechanism. The indicator moves on a scale calibrated in units of pressure (**Fig. 2**). They have a heavy glass window and a safety flap on the back. If the Bourdon's tube ruptures, then the gas will be vented from the back of the case rather than blowing out the glass window in the front. The full scale pressure indication is at least 33% greater than the maximum cylinder pressure. The pressure gauges are color coded and also identified by the name or symbol of the gas being measured. They must be positioned in such a way that they are easily visible and not covered by other items.

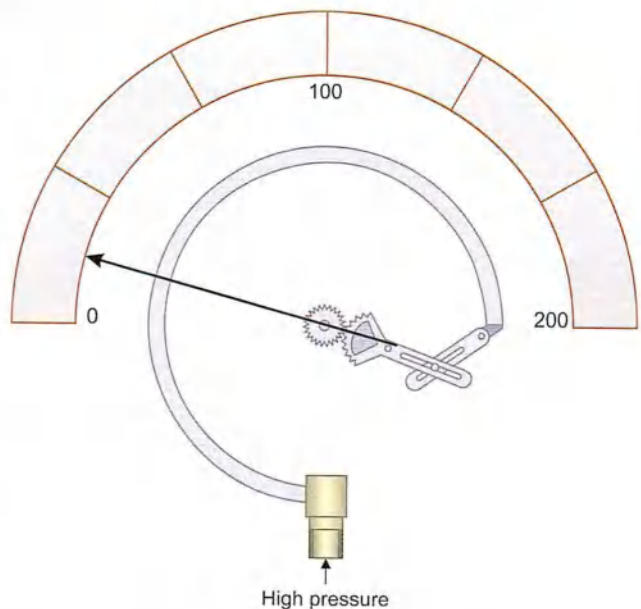


Fig. 2: Bourdon pressure gauge.

Q. Why do you need pressure regulators?

Ans.

- The pressure reducing valves reduce the variable high pressure in the cylinder to a constant low pressure suitable for use in the anesthesia machine. The cylinder pressures are reduced to 375 kPa (45 psi):
 - When a cylinder is being used the pressure decreases due to which the flow also reduces. As a result, the flow control valve has to be frequently adjusted to maintain a steady flow. Pressure regulators were used to ensure constant flow with changing supply pressure thus eliminating the need for frequent adjustments in the flowmeter.
- When there is high pressure it is difficult to make small adjustments in the flowmeter. By lowering the pressure, it allows fine adjustments to be made easily.
- Also lowering the pressure reduces the possibility of dangerous pressure build up which can damage the anesthesia machine.

Q. What is the working principle of a pressure regulator?

Ans. The pressure regulator has a high pressure chamber with an inlet that has a filter. The high pressure chamber has a valve which leads to a low pressure chamber with an outlet. The low pressure chamber has a diaphragm attached to a spring. The gases from the cylinders pass through the inlet into a high pressure chamber. The gases then pass through the valve into the low pressure chamber. The increased pressure in the low pressure chamber tends to distend the diaphragm. The distension of the diaphragm is opposed by the tension in the spring (S). When there is sufficient increase in pressure the diaphragm moves and the valve closes. The pressure at which the valve closes can be preset. This is done by adjusting the screw which alters the tension in the spring. When the gas is allowed to flow through the outlet, there is a drop in pressure in the low pressure chamber and the valve opens. If the outlet is closed the pressure again builds up and the valve closes. Thus the force exerted by the high pressure gases tries to close the valve while the opposing force of the diaphragm and spring tries to open the valve. The balance between the two opposing forces helps to maintain a constant gas flow (**Fig. 3**).

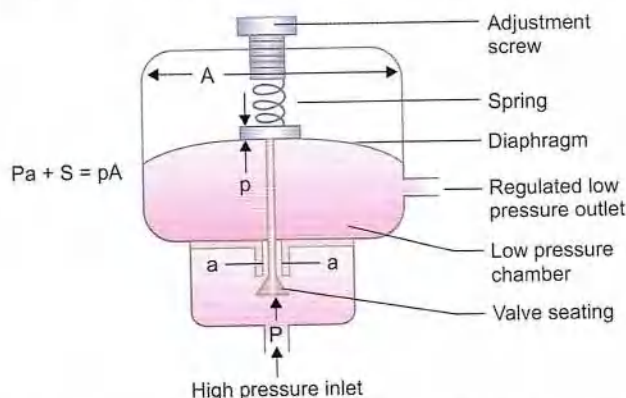


Fig. 3: Pressure regulator. Pressure is defined as force acting against an area. The force can be increased by increasing the pressure or by increasing the area over which the pressure acts. In a pressure regulator, large pressure (P) acting over a small area (a) is balanced by smaller pressure (p) acting over a large area (A).

Q. What is Adams valve?

Ans. Adams valve is a pressure-regulating valve which was used in the past. The working principle is the same as the newer pressure regulators, but the internal structure is different. Instead of the push rod there is lazy tongs toggle arrangement. It reverses the direction of thrust transmitted from the diaphragm.

Q. Why do you have fins on the Adams regulator for nitrous oxide?

Ans. Earlier nitrous oxide contained significant amounts of water vapor as impurity. When nitrous oxide was being

used, the cooling of the valve caused condensation of the water vapor on the valve seating. When there was excessive cooling the water vapor used to freeze and jam the valve. The fins on the nitrous oxide regulator helped in conducting heat from the surrounding. This prevented excessive cooling of the valve which in turn prevented the freezing of water vapor. Modern pressure regulators do not have fins as nitrous oxide does not have water vapor as impurity.

Q. What are the safety features on pressure regulators?

Ans. Pressure regulators have safety relief valves downstream of the regulators. If due to any reason there is build up in the pressure regulator, then the safety valve blows off at a set pressure of 525 kPa (70 psi). The spring-loaded valves close when the pressure reduces. Some valves may rupture and remain open till they are repaired. The safety feature is not present in Boyle's Machine but present in newer machines.

The older pressure regulators were coded and labeled for specific gases.

Q. What is the normal working pressure in the anesthesia gas machine and cylinders?

Ans. The hospital pipeline is the primary source of all gases and the pressure within the pipelines is 60 psig (50 psig in US), which is the normal working pressure of most machines. The oxygen cylinder pressure is 2,000 psi which is regulated to approximately 45 psi after it enters the machine. A full nitrous oxide cylinder pressure is regulated from 745 psi to approximately 45 psig. Air cylinder pressures are similar to oxygen.

Q. Why is the regulated cylinder pressure lower than the pipeline pressure?

Ans. The pipeline pressures are 55–60 psi and the regulated cylinder pressures are 45 psi. Regulating the cylinder pressure below the pipeline pressure reduces the risk of premature emptying of cylinders if the cylinders are accidentally left open. Even if the cylinder is left open the pipeline gas will be preferentially used.

Q. What are the safety features in the pipeline system?

Ans. The terminal wall units for the pipelines have Schrader socket assembly which accepts the Schrader probe for a specific gas. The terminal wall units are color coded and the identity of the gas is also mentioned on the socket. The socket assembly has a primary valve (automatic shutoff valve) which allows the gas to flow when the pipeline is inserted and which closes automatically when the pipeline is disconnected from the wall outlet. The socket assembly also has an isolating valve which prevents any gas leak when the primary valve is removed for cleaning or replacement.

The pipeline system consists of flexible noncrushable hoses which connect the terminal wall outlets to the anesthesia machine. The pipeline has a Schrader probe

which fits into the Schrader socket in the terminal wall outlet. Though the size of the Schrader probe is the same for all gases, the probes have a collar indexing system specific for a gas. The collar for each gas has a unique diameter which will fit the matching recess on the Schrader socket assembly for that gas. Nowadays instead of Schrader probes there are quick connectors which are gas specific and manufacturer specific. The quick connectors on the wall outlet will not accept pipeline Schrader probe. The pipeline tubing's are also color coded.

The machine end of the pipelines has the diameter index safety system (DISS) or noninterchangeable screw thread system (NIST) to minimize the risk of misconnections. The connections for oxygen, nitrous oxide, and air have different diameters.

There are check valves at the pipeline inlets to the machine, which prevent the backflow of gas from the machine to the pipelines or to the atmosphere when pipelines are not attached to the machine.

Q. How will you shutoff the flow of gases if there is leakage from the wall outlet?

Ans. If there is any need for maintenance, repair, testing or expansion then the shutoff valves can be used to isolate specific areas of the pipeline system. These valves are of two types. The manual shutoff valves are easily visible and can be accessed at all times. The service shutoff valves are secured and locked to prevent accidental closing and can be accessed only by authorized personnel. For all the pipelines entering the operation theaters, there is a shutoff valve (bypass valve) just outside the operation theaters. If there is any gas leakage from the wall outlet or the pendants, then this bypass valve can be used to shut off the gas supply to the operation theaters (Fig. 4).



Fig. 4: Shutoff valve.
(VCC: vapor compression cycle)

Q. What should be the ideal position of pipeline pressure gauge?

Ans. The pipeline pressure gauge should ideally be on the pipeline side of the check valve. If it is on the machine side

of the check valve it will detect the machine pressure and not the pipeline pressures.

Q. How would you know what is the position of the pipeline pressure gauge?

Ans. The pipelines should be disconnected and the cylinders should be opened. If the pipeline pressure gauge reads zero, then the pressure gauge is on the pipeline side. If there is a reading, then it is on the machine side.

Q. Why do some machines have secondary pressure regulators?

Ans. Some machines have secondary pressure regulators in the intermediate pressure system just upstream of the flowmeters. They receive the gas from the pipeline or cylinder regulator and reduce it to 14 psig for oxygen and 26 psig for nitrous oxide. The use of ventilators and oxygen flush can sometimes cause sudden pressure fluctuations which can affect the performance of the flowmeters. A secondary pressure regulator helps to minimize these pressure fluctuations. The flowmeter settings remain constant as the pressure is reduced below the normal fluctuation range. Thus oxygen flow from the flowmeter will remain constant as long as the oxygen pressure is above 14 psig.

Also the mechanically linked antihypoxia devices at the flowmeter level need a constant oxygen supply pressure to maintain accurate gas flows. However, these devices cannot detect altered gas flow rates caused by changing pressures. Whenever the oxygen supply pressure decreases below 20 psig the pressure sensor shutoff valve cuts off supply of all the other gases. Due to the secondary pressure regulators in Datex Ohmeda machines the pressure of oxygen is lower than that of nitrous oxide which ensures that oxygen would be the last gas flow to decrease.

Q. What are the flows and pressure at which oxygen is delivered from the oxygen flush valve?

Ans. Oxygen flush valve receives oxygen from the pipeline inlet or oxygen cylinders at a pressure of 45–55 psi and delivers it to the common gas outlet at flow rates of 35–75 L/min. In the older machines, the oxygen flush valve had a locking mechanism. This can cause barotrauma if the valve remains in the locked position. In the modern machines, the oxygen flush button is in a recess to prevent accidental activation and there is no locking mechanism.

It bypasses the flowmeter and vaporizers.

Q. What are the hazards of oxygen flush valve?

Ans. There are several hazards associated with the oxygen flush valve:

- Barotrauma can result from sticking of defective or damaged valve in the fully open position. In valves with locking mechanism, accidental locking of the valve can

cause barotraumas. The oxygen flush valves on modern machines do not have locking mechanism.

- If the oxygen flush valve is incompetent, then the oxygen flowing through the valve can dilute the inhaled anesthetic resulting in awareness in the patient. Repeated use of oxygen flush intraoperatively can also dilute the inhaled anesthetic.
- If the anesthesia machine does not use fresh gas decoupling or inspiratory pressure limiter, then the use of oxygen flush during the inspiratory phase of positive pressure ventilation can cause barotrauma in patients. In the traditional machines, the ventilators relief valve is closed and APL valve is out of circuit during controlled ventilation, due to which the excess gas cannot be vented out during the inspiratory phase of controlled ventilation.
- If the vaporizers are placed downstream of the common gas outlet, then the use of oxygen flush can deliver large quantities of inhaled anesthetic to the patient.
- Oxygen flush should not be used to detect leaks in the low pressure circuit in anesthesia machines with one-way check valve at the common gas outlet. Major leaks in the low pressure system can go undetected because the back pressure from the breathing system closes the one-way valve making it air tight.

Q. What are the safety devices for oxygen supply pressure failure?

Ans. These devices prevent the delivery of hypoxic mixture resulting from a decrease in oxygen supply pressure. They are fail safe valve which are present in the gas line supplying all the flowmeters except that for oxygen. In the older machines, whenever the oxygen supply pressure decreases below 50% of the normal supply pressure or below 30 psi the valve shuts off or proportionately decreases the supply pressure of all the other gases. This is also called as *master and slave mechanism* where oxygen is the master and nitrous oxide is the slave. Here the tension in spring of the nitrous oxide pressure reducing valve is replaced by the pressure of oxygen coming from the oxygen pressure regulating valve. If the oxygen pressures drop below 30 psi, the valve in the nitrous oxide pressure regulator closes and no further nitrous oxide can be delivered.

In Datex Ohmeda machine there is the pressure sensors shutoff valve with a threshold pressure of 20 psig. Whenever the oxygen supply pressure falls below 20 psig the valve closes and completely stops the supply of nitrous oxide to the flow control valve.

In Dräger machines, there is the oxygen failure protection device (OFPD). This device is based on the proportioning principle and not on the threshold principle. Whenever there is a decrease in the oxygen supply pressure there is a proportional decrease in pressure of all the gases controlled

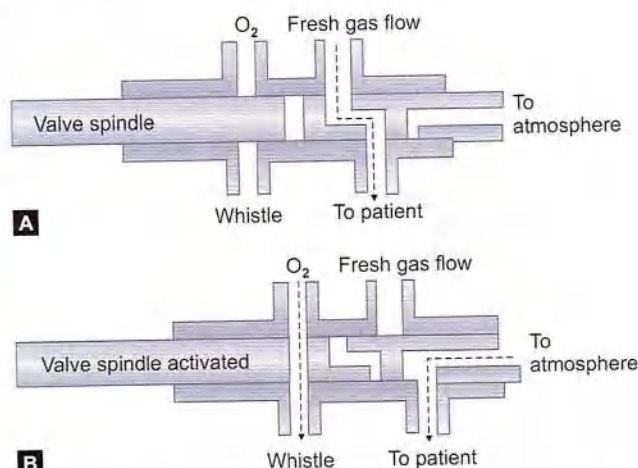
by the OFPD. When the oxygen supply pressure falls below 10 psig the supply of other gases to the respective flowmeters is completely cut off.

The Fabius GS Dräger machine, uses a Sensitive Oxygen Ratio Controller (S-ORC) shuts off nitrous oxide if the oxygen flow is <200 mL/min, or if the oxygen fresh gas valve is closed.

Q. When is the oxygen pressure failure warning device activated?

Ans. Ritchie whistle is an oxygen failure warning device. It was introduced in the mid-1960s. Anesthetic gases will flow into the machine as long as the pipeline pressure is 55–60 psig. When the pressure drops below 260 kPa (38 psig) the anesthetic gas cutoff valve begins to close and there is an audible alarm (**Figs. 5A and B**). At a pressure of 200 kPa (30 psig) the anesthetic gases are cut off. The alarm continues till the oxygen pressure falls to 40.4 kPa (6 psig). However, there is a mechanism which allows the patient to inspire room air.

In Fabius GS machine, there is an audible and visual alarm when the oxygen pipeline pressure drops below 20 ± 4 psi.



Figs. 5A and B: (A) Oxygen failure warning device and (B) When oxygen supply pressure falls below 200 kPa then whistle is activated.

Q. What are the limitations of the oxygen pressure failure safety devices?

Ans. The oxygen pressure failure safety devices depend on pressure and not on flows. Therefore, machines that do not have a flow proportioning system can still deliver hypoxic mixture. These devices only prevent hypoxia due to problems occurring upstream of them, e.g. disconnected oxygen hoses, low oxygen pressures in the pipelines, and depletion of oxygen cylinders. They do not protect against hypoxic mixtures from being delivered due problems downstream of these devices. Hypoxic mixture can result from problems such as crossover in the pipelines, wrong

contents in the gas cylinder, leaks in the equipment, and operation errors such as inadvertently closed oxygen flow control valve. In these situations, the normal oxygen supply pressure keeps the other gas lines open.

Q. What mechanisms are incorporated in an anesthetic machine to prevent delivery of a hypoxic mixture?

Ans. There are various devices that can help prevent the delivery of hypoxic mixture.

Minimum mandatory oxygen flow: In some anesthesia machines there should be a minimum oxygen flow of 50–250 mL/min. before other gases can flow. This is achieved by providing a mechanical stop on the oxygen flow control valve or a resistor which allows a small flow to bypass the oxygen flow control valve. However, minimum oxygen flow by itself will not prevent the delivery of hypoxic mixtures.

The minimum oxygen ratio or the proportioning devices prevent the delivery of hypoxic mixture. Oxygen and nitrous oxide are linked either pneumatically or mechanically to ensure a minimum oxygen concentration of 23–25% at the common gas outlet. Therefore, the flowmeters are adjacent to each other. The Datex Ohmeda has Link-25 Proportion-Limiting Control System while Drager has oxygen ratio monitor controller (ORMC). Newer Drager machines have sensitive oxygen ratio controller (S-ORC).

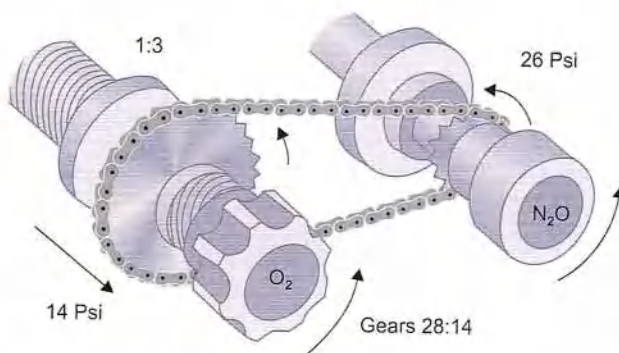


Fig. 6: Datex Ohmeda Link-25 system.

Mechanical devices: In the Datex Ohmeda Link-25 system a 28-tooth sprocket is attached to the oxygen flow control valve and 14-tooth sprocket is attached to the nitrous oxide flow control valve. The sprockets are linked by a chain with a gear ratio of 2:1 (nitrous oxide: oxygen). As a result of this when nitrous oxide flow control valve turns two revolutions; oxygen flow control valve turns one revolution. A second stage pressure regulator reduces the pressure at the nitrous oxide flow control valve to 26 psig and at the oxygen flow control valve to 14 psig. So the final flow ratio is 3:1 (**Fig. 6**). The final oxygen concentration is a result of this combination of mechanical and pneumatic aspects of the system. This

system actually increases the flow of oxygen to prevent the delivery of a fresh gas mixture with an oxygen concentration of <25%. Whenever nitrous oxide is flowing the oxygen flow control knob can be opened independently, but it cannot be closed below a setting which will result in <25% oxygen in the mixture. Another safety feature of this system is that a mechanical stop on the oxygen flow control valve ensures a minimum flow of 175–250 mL of oxygen even when the valve is closed.

Pneumatic devices: In the Drager machines the ORMC and S-ORC are pneumatic oxygen-nitrous oxide interlock system which ensure a fresh gas oxygen concentration of at least 25% + 3%. They actually limit the flow of nitrous oxide to prevent the delivery of hypoxic mixture. This system has an oxygen chamber, a nitrous oxide chamber, and nitrous oxide slave control valve which are all interconnected by a horizontal shaft. The slave control valve consists of a spring-loaded ball which is linked to O₂ diaphragm. It cuts off flow across the N₂O needle on the base of its flowmeter, if O₂ supply fails or if O₂ flowmeter is turned off. The oxygen and nitrous oxide flow control valves have resistors downstream. Back pressure generated due to flow of gases is transferred to the oxygen and nitrous oxide chambers. There is a diaphragm interposed between the two chambers. The relative values of the resistors determine the value of the controlled fresh gas oxygen concentration. The value of the oxygen flow tube resistor is 3–4 times that of the nitrous oxide flow tube resistor. Back pressure in the oxygen and nitrous oxide chambers cause movement of a horizontal shaft attached to the rubber diaphragm. The movement of the shaft controls the nitrous oxide slave control valve which feeds the nitrous oxide flow control valve:

- **O₂ flowmeter turned on:** It pushes the shaft to the left thus pushing the ball away from the N₂O needle. N₂O is thus ready to be turned on.
- **O₂ flow turned off:** The shaft moves to the right to return to its neutral position, this allows the spring on the ball to recoil and close the N₂O needle. N₂O cannot be turned on.
- **If one attempts to increase the N₂O flow:** Pressure in the N₂O chamber increases and moves the shaft to the right, thus preventing its increase. As the shaft moves to the left, the spring relaxes and the ball partly occludes the flow from the N₂O needle.
- **If one attempts to reduce O₂ flow:** Pressure in the O₂ chamber decreases and the shaft moves to the right, which reduces N₂O flow from the needle.

In this system, if one tries to increase N₂O flow, they will not be able to do so as the flow of N₂O depends on O₂ flow.

Electronic devices: In Penlon machines, there is a paramagnetic oxygen analyzer. Whenever the oxygen concentration is <25% there is an audible alarm and nitrous oxide supply is cut off.

Q. Can these systems prevent delivery of hypoxic mixture to the patient?

Ans. No. These systems work only for two gases namely O_2 and N_2O . Third or fourth gases such as air or helium are not linked. In addition, these systems prevent hypoxic mixture only until the common gas outlet. If a circle system is used, inappropriately low flow settings can lead to hypoxic mixture within the circle system.

Q. What are the circumstances in which a hypoxic mixture can be delivered even when the hypoxic guard system is used?

Ans.

- Wrong supply gas in oxygen pipeline or cylinder.
- Defective pneumatics or mechanics (the hypoxic guard system is broken).
- Leaks downstream of flowmeter control valves.
- As the proportioning system links only oxygen and nitrous oxide the administration of a third inert gas such as helium, nitrogen or carbon dioxide can cause a hypoxic mixture.
- The hypoxic guard system only connects oxygen and nitrous oxide. The volatile inhaled anesthetics are added to the gas mixture downstream from the flowmeters and proportioning system. Hence, when using high concentration of low potency inhaled anesthetics (maximum dial setting for desflurane is 18%), it is possible to create a hypoxic mixture when you give desflurane in air.

Therefore, it is essential to monitor the inspired concentration of oxygen being delivered from the machine outlet.

Q. How would you describe a flowmeter assembly?

Ans. The flowmeter assembly has a needle valve, valve seat, and tapered glass tube which is calibrated for the particular gas. The flowmeter is made up of glass and is made gas tight at both the ends by "O" rings and washer. Each flowmeter is calibrated for the specific gas at 20°C at ambient pressure of 760 mm Hg.

The flowmeters are designed to deliver a range of flows from a single tube. The bore of the tube increases from the bottom to the top. This is achieved by either single tapered or double tapered tubes. Some anesthetic machines have a pair of tubes for each gas: one to be used for low flows and the other for higher flows. As the pair is arranged in series, the second tube indicates total flow. It usually contains a bobbin or ball which is free to rotate and to move up or down depending upon the flow rate.

Q. How do you read flows from flowmeters with bobbins?

Ans. The flows are read off from the top end of the bobbin. A freely rotating bobbin confirms that the flowmeter is delivering the gases at the flow rate indicated. They have grooves cut at an angle which force it to rotate as the gas flows from the lower end of the tube. A mark on the bobbin aids in visual confirmation that it is rotating.

Some flowmeters such as those attached to cylinders in the ward or wall mounted flowmeters have a sphere as the bobbin within them. The flow rate reading is taken from the equator of the sphere.

Q. How does gas flow around the bobbin?

Ans. At low flows, the gap between the flange of the bobbin and the flowmeter wall behaves like a tube (distance between the flange and the wall is small compared to the length of the flange). Therefore, gas flow depends on viscosity. At high flows, the bobbin is pushed up the flowmeter tube. Here the gap between the flange and the wall of the flowmeter will behave like an orifice (distance between the flange and the wall is great when compared to the length of the flange). Therefore, gas flow depends on density.

Q. What are the principles behind the flowmeters?

Ans. It is a variable orifice (area)/constant pressure flowmeter. The variable area is accounted by the tapered flowmeter tubes. As the bobbin moves upward the area or space around it increases therefore variable area (orifice). If the pressure across (above and below) the bobbin is measured, it is always constant, and hence the constant pressure flowmeter (**Fig. 7**).

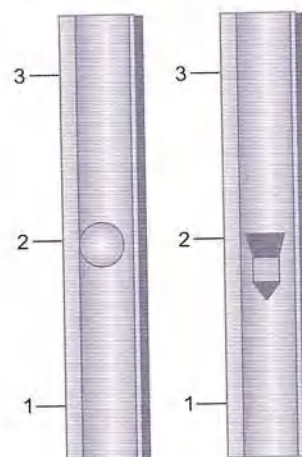


Fig. 7: Functioning of flowmeter.

The downward force on the bobbin is due to the force of gravity acting on its mass. This can be assumed to be constant for the entire height of the flowmeter (as it is very short). Therefore, the force needed to balance (by gases flowing from the bottom of the flowmeter) the bobbin against its weight will also be constant.

Q. What factors may contribute to erroneous reading of a flowmeter?

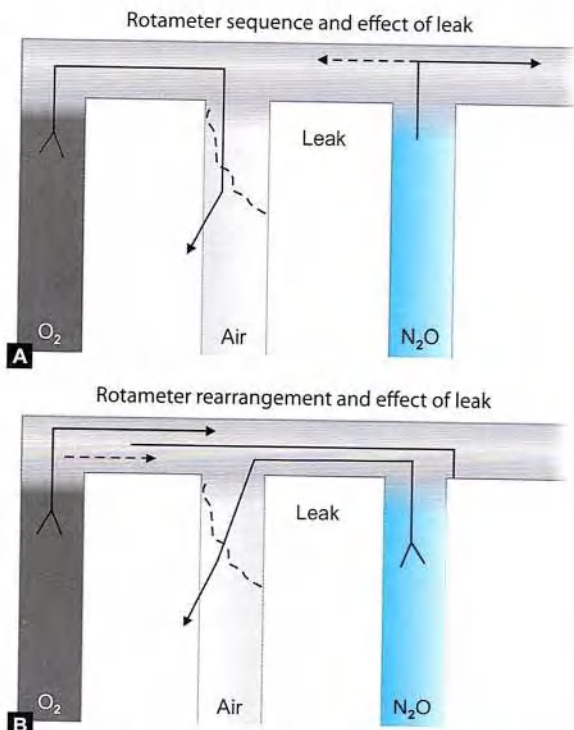
Ans. Spinning of the bobbin is essential for accurate reading. The bobbin may stick to the flowmeter tube under the following circumstances:

- Dust in the flowmeter—filter is incorporated at the needle valve at the base of the flowmeter to trap any dust.
- Static electricity—the glass is coated with a conductive material (gold or tin oxide) to prevent building up of static electricity.
- Tilting of the flowmeter at its mounting—not significant in a modern anesthetic machine.
- Back pressure from a ventilator—the bobbin under-reads, but the actual flow is not significantly altered.
- Translocation of flowmeters after servicing—flowmeters are color coded; the symbol of the gas is etched on it and indexed at the needle valve thus reducing risk of translocation.

Q. How are the flowmeters arranged in an anesthetic machine?

Ans. Conventionally, from left to right it is O_2 , N_2O , and air. O_2 is carried by a separate line on top of the flowmeter block and it is the last gas to join the back bar. For example, let us consider the following scenarios:

Scenario 1: The anesthetist is using O_2 and N_2O a crack develops in the air flowmeter which is switched off, O_2 will flow out through this crack preferentially and a hypoxic mixture will be delivered at the common gas outlet (**Figs. 8A and B**).



Figs. 8A and B: Arrangement of flowmeter in anesthetic machine.

Scenario 2: Rearrangement of the flowmeter block—the anesthetist is using O_2 and N_2O , a crack develops in the air flowmeter which is switched off, N_2O will be vented off through the crack and as oxygen is the last gas to enter the gas mixture, thus preventing delivery of hypoxic gas.

Q. What safety measures are built into the flowmeter block?

- All the needle valve control knobs are color coded and clearly marked.
- O_2 control knob is larger, octagonal profile, and protrudes out more than nitrous oxide knob when completely closed (**Fig. 9**).



Fig. 9: Color-coded knobs in flowmeter.

- The torque required to turn the knobs is high enough to prevent adjustments secondary to casual contact.
- Some manufacturers use a plastic shield to cover the knobs to prevent alterations in flow settings on casual contact.
- Illuminated back helps visualization of the flowmeter block in dim light.
- Sequence of various gases to minimize delivery of hypoxic mixture.

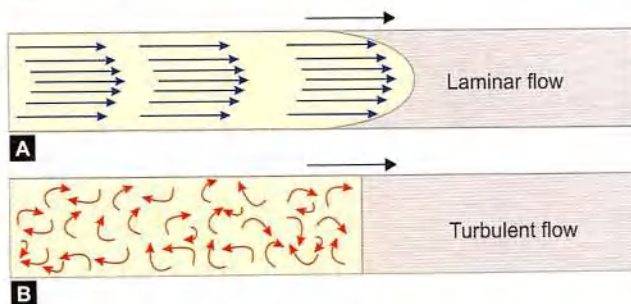
Q. How do electronic flowmeters work?

Ans. In S/5 Datex machines: A chamber collects gases that pass through the needle valve. As the volume of the chamber is fixed, the pressure within it increases. The chamber is controlled by a solenoid valve that opens and lets the gas out when a specific pressure limit is reached. The number of times the solenoid valves opens per minute is related to flow per minute. The display is either numerical (Drager) or as a bar (DatexS5). Electronic flowmeters allow data to be stored and printed off as a part of electronic anesthetic chart.

Q. Define flow. What do you understand by laminar and turbulent flow?

Ans. Flow is defined as the quantity of fluid (gas or liquid) passing a point in unit time.

Laminar flow: The fluid moves in a steady manner and there is no turbulence or eddy current. The flow is greatest in the central column (twice the mean flow) laterally the flow is slower compared to the center until approaches zero at the sides. It is present in smooth tubes at lower flow rates (**Figs. 10A and B**).



Figs. 10A and B: (A) Laminar flow and (B) Turbulent flow.

Laminar flow is directly proportional to pressure gradient across the tube and the radius of the tube and indirectly proportional to the viscosity and the length of the tube. So decreasing the radius by half decrease the flow by a factor of 16 and halving the length doubles the flow. This is explained by the Hagen-Poiseuille equation:

$$Q \propto \frac{\Delta P r^4}{8 \eta l}$$

Where, Q is the flow, ΔP is the pressure gradient across the tube, r is the radius of the tube, η is the viscosity, and l is the length of the tube.

Anesthetic implication: During fluid resuscitation a short length-wide bore cannula is preferred, e.g., 14 G as the flow rate is much superior to a 20-G cannula or a central line.

Similarly, intubating patients with a very small tube increases the resistance to flow and thus the pressure increases to deliver the same amount of flow through the endotracheal tube.

Turbulent flow: Turbulent flow is when there are eddies in the fluid. It is seen in the following conditions:

- Rough or irregular inner surface of the tube
- Acute bends or acute narrowing in the tube
- Flow velocity exceeds a critical level.

Q. What is Reynolds number?

Ans. Reynolds number is,

$$Re = v\rho D/\eta$$

Where, v —velocity of the fluid, ρ —density of the fluid, η —viscosity, and D —diameter of the tube.

A Reynolds number in excess of 2,000 favors turbulent flow. If it is <2,000 then laminar flow exists. The density of the fluid is in the numerator, hence a fall in the density would reduce Reynolds number and so encourage laminar flow.

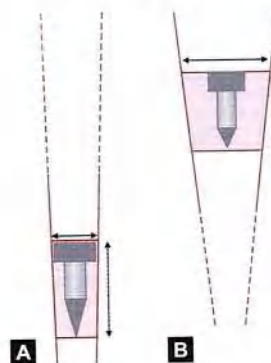
Q. How is this clinically important?

Ans. In cases of upper airway obstruction, there is turbulent flow which increases the work of breathing. Administering helium, which has very low density decreases the Reynolds number and thus changes the characteristics of flow from turbulent to laminar and decreases the respiratory work.

This is the reason behind using heliox to reduce respiratory distress due to upper airway obstruction.

Q. Can the flowmeter for different gases be used interchangeably?

Ans. No. A flowmeter is tapered. At low flows, the gap between the walls of the flowmeter and the bobbin behaves like a tube so viscosity is important. Viscosity is independent of barometric pressure so at low flows it reads accurately. At high flows, the gap between the bobbin and the walls of the flowmeter behave like an orifice and the flow is dependent upon the density of the gases. As different gases have different densities the flowmeters are calibrated for a specific gas and cannot be used interchangeably (**Figs. 11A and B**).



Figs. 11A and B: Uses of flowmeter for different gases. (A) Low flow rate and (B) High flow rate.

Q. Can the same flowmeters be used at different altitudes?

Ans. No. Barometric pressures affect the density but do not affect viscosity of the gas. At high flow rates the flow is dependent upon the density of the gases. At high altitude, density decreases, so the flowmeter under reads at high flows. In a hyperbaric chamber it over-reads at high flows.

■ CIRCLE SYSTEM

Q. What are the components in a circle system?

Ans. See **Figure 12** for components in a circle system.

Although there are many possible arrangements of the components, in practice the arrangement is dictated by certain rules and practicalities.

Fresh gas flow (FGF): Ideally located downstream to the soda lime canister and upstream to the inspiratory one-way valve. During expiratory pause continuous FGF pushes the CO_2 -enriched exhaled gases retrograde past the soda lime canister to be vented via the adjustable pressure-limiting (APL) valve. This prolongs the life of soda lime.

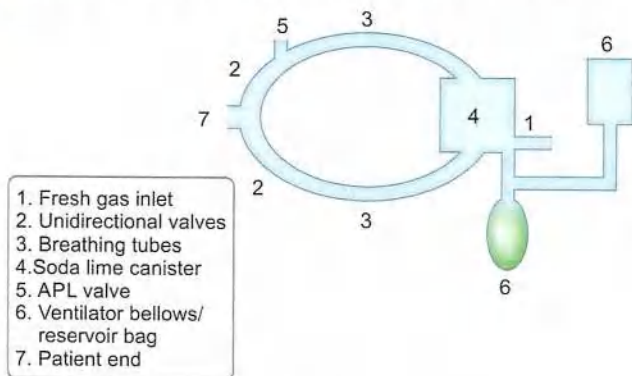


Fig. 12: Circle system.
(APL: adjustable pressure-limiting)

If FGF is high, it flushes out gases that have been cleaned up by soda lime, and if high enough, it might be lost via the APL valve. In addition, changes in FGF composition is rapidly reflected in the inspired gases.

Fresh gas flow joining upstream of the soda lime has the following disadvantages:

- Its composition may not immediately reflect the gas content of inspired gases.
- Activation of flush may carry dust.
- Inhaled anesthetic agent is absorbed by soda lime.
- High flows may dry up soda lime.

If FGF enters between the patient and the expiratory unidirectional valve, there is a risk of barotrauma on activation of the O₂ flush at a critical time of respiratory cycle.

Unidirectional Valves

These are light disk sitting on a knife edge seat. The disk is held in place by a cage. The valve is designed to prevent the disk from sticking to valve seat. Gases normally flow from under the disk, lifting the disk off the seat and flowing out under the dome. Pressure from under the dome firmly seats the disk and prevents retrograde flow (**Fig. 13**). Some unidirectional valves are positional and depend on gravity for seating the disk. Historically, valves were placed on a block near the patient end.

Reservoir bag: Commonly between the expiratory valve and the canister. This reduces the work of expiration which is the only work the respiratory muscles do under intermittent positive pressure ventilation (IPPV). If it is located between absorber and inspiratory valve, it reduces work of inspiration in spontaneous ventilation. Any positions between the patient and the unidirectional valves are not permitted as it would lead to rebreathing.

Adjustable pressure-limiting valve: It is usually located between the expiratory unidirectional valve and the canister. This allows exhaled gases to escape before passing through the soda lime canister. Downstream the canister will lead to loss of gases that are conditioned (FGF

and gas from the canister). Downstream to inspiratory unidirectional valve leads to rebreathing, in addition to loss of conditioned gases.

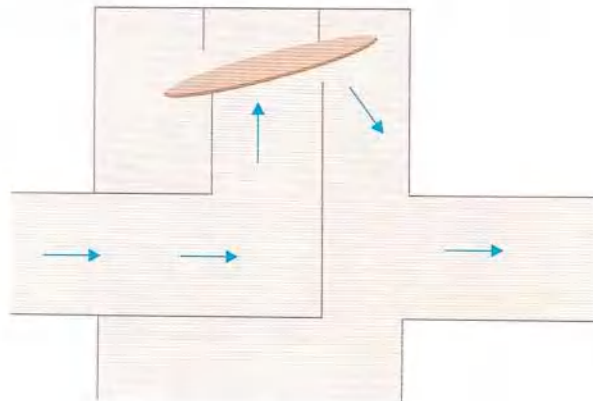


Fig. 13: Unidirectional valves.

Other Components in the Circle Housing

A selector switch toggles between ventilator and the reservoir bag. In newer machines the APL valve is automatically isolated (along with the bag) when switched to ventilator mode.

Positive end-expiratory pressure (PEEP) valve may be added by replacing the dome of the expiratory port. In newer machines it is incorporated in the dome of the expiratory unidirectional valve.

Gases aspirated for analysis are returned to the circle system by a port that is functionally upstream of the canister. The APL valve is connected to a scavenging system.

A disposable circle system is manufactured by intersurgical which is useful in infective cases and patients who are susceptible to malignant hyperpyrexia.

Q. What are the advantages and disadvantages of a circle system?

Ans.

Advantages:

- Economical:
 - Gases and inhalational anesthetic agents
 - Scavenging volume/load decreases.
- Better preservation of heat and humidity within the circuit.
- Arterial CO₂ tension depends on minute ventilation and not on FGF.
- Low dead space if no faults in the system. The dead space in the circle system consists of the Y-piece which connects the inspiratory and the expiratory limb.
- Reduced atmospheric pollution.

Disadvantages:

- Many components and therefore there is a risk of disconnection and misconnection.

- Slow change in the inspired gas composition particularly with vaporizer-out-of-circuit and low flow. Change in the composition of inspired gases requires the FGF rate to be increased.
- Dry soda lime or Baralyme adsorbs anesthetic agents.
- Accumulation of trace gases—carbon monoxide (CO), H₂, acetone, methane, and ethanol. CO comes from smokers and from breakdown of Hb.
- Acrylic monomer is exhaled after cementing. A brief period of higher FGF would vent this out.
- Greater resistance to breathing:
 - Position of the bag influences the resistance to that part of respiratory cycle, e.g., in a patient breathing spontaneously, if the bag is in the inspiratory limb, it reduces inspiratory work of breathing.
 - A high FGF reduces inspiratory resistance but increases expiratory resistance.

Q. How is CO₂ absorbed in the circle system?

Ans. The principle of CO₂ absorption is base neutralizing an acid. Carbon dioxide reacts with water to form carbonic acid which is neutralized by hydroxides of alkali. This reaction takes place between ions; hence water has to be present so that the acid and base can exist in the ionic form. CO₂ combines with water to form H₂CO₃ which is neutralized by Na or CaOH. The final reaction generates water and a carbonate.

Q. What is the composition of CO₂ absorbents?

Ans. There are two commercial preparations namely soda lime and barium lime. Soda lime is made up of approximately 80% calcium hydroxide, 4% sodium hydroxide, and 15–20% water. Sodium hydroxide has hygroscopic properties and improves the reactivity of the mixture. Absorbents with low water content would exhaust rapidly, while those with high water content would get sticky and provide increased resistance for gas flow. Small amounts of silica and kieselguhr are added as hardeners. They help to form the required granule size. They are no longer used in the modern manufacturing process. KOH is also no longer added. Indicators are also present.

Barium lime is made of 80% calcium hydroxide and 20% barium hydroxide. Barium hydroxide has its own naturally occurring water of crystallization. It does not require any hardeners as water of crystallization prevents dust formation. Indicator is present.

Q. Why are hardeners added to soda lime?

Ans. Soda lime granules powder very easily to produce fine dust. If there is excessive powder, it can cause channeling, caking, and increased resistance to flow. If the dust is blown into the patient, it can irritate the respiratory mucosa. Small amount of silica is added to soda lime to increase its

hardness. Kieselguhr is added to silica as it tends to clog the pores and reduce the efficiency of soda lime.

Q. What should be the hardness number and how is it tested?

Ans. Hardness is tested by agitating a weighted amount of absorbent with a steel ball and agitating it. The soda lime is then sifted through eight mesh screen; the amount of soda lime in percentage left on the mesh screen is the hardness number. Normally, it should be greater than 75.

Q. Which is the more active component in soda lime?

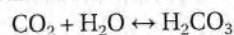
Ans. Sodium hydroxide is the more active component in soda lime.

Q. Why do you have only 4% sodium hydroxide and 80% calcium hydroxide in soda lime?

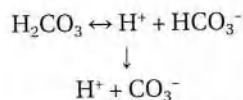
Ans. Sodium hydroxide is more hygroscopic. If you increase the concentration of sodium hydroxide, then it will absorb water whenever there is high humidity. This will result in clogging of the pores of the granules and caking during use. Also lot of heat is produced during this reaction.

Q. What is the chemical reaction taking place during CO₂ absorption by soda lime?

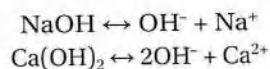
Ans. Carbon dioxide first reacts with the water on the surface of the soda lime granules to form carbonic acid:



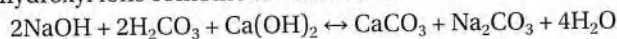
As it is a weak acid, it incompletely dissociates into its ions:



Sodium and calcium hydroxide also dissociate into their ions:



The sodium and calcium ions combine with carbonate ions to form sodium and calcium carbonate. Hydrogen and hydroxyl ions combine to form water:



This is an exothermic reaction which produces a lot of heat.

Q. Is the size and shape of soda lime granules important?

Ans. The size and shape of granules are important. Irregular surface of the granules provides maximum surface area for absorption. Also if the granules are smaller in size they provide greater surface area for absorption and decrease channeling. But there is increased resistance and caking with smaller granules. If the granules are large they provide lesser resistance to air flow but have lesser surface area for absorption. The optimum size of the granules is 1.5–5 mm or 4–8 mesh size (USA) and 3–10 (UK). A size-4

mesh means the sieve has squares of 1/4 inch and size 8 means there are squares of 1/8 inch. Thus 4-mesh size is bigger than 8-mesh.

Q. How do you know that soda lime is exhausted?

Ans. Indicators are added to the absorbent to indicate that they are exhausted. These indicators are acids or bases and their color depends on hydrogen ion concentration. Various indicators are available; these are summarized in Table 5.

TABLE 5: Exhausted of soda lime through various indicators.

Indicator	Color when fresh	Color when exhausted
Phenolphthalein	White	Pink
Ethyl violet	White	Purple
Clayton yellow	Deep pink	Off white
Ethyl orange	Orange	Yellow
Mimosa Z	Red	White

Q. What are the absorptive capacities of CO₂ absorbents?

Ans. The absorptive capacity of soda lime is 25 L of CO₂ per 100 g and of barium lime is 27 L of CO₂ per 100 g of absorbent. However, during continuous use the outer surface of the granule is exhausted before the whole granule is used up. Therefore, it appears that the soda lime is exhausted even before its absorptive capacity is reached. Hence, during use the 500 g soda lime canister appear exhausted at a CO load of 10–12 L per 100 g and the 2 kg jumbo canister appear exhausted at a CO₂ load of 17 L per 100 g.

Q. Why does soda lime which is exhausted show reversal of color when allowed to rest?

Ans. The reversal of color is due to regeneration of soda lime. The length of the rest period will determine the amount of regeneration. Sodium hydroxide is more active than calcium hydroxide. So it preferentially binds to carbon dioxide to form sodium carbonate. As sodium carbonate is more soluble, it dissolves in the moisture on the granules and penetrates into the granules. Within the granules it reacts with calcium hydroxide to form calcium carbonate (which is insoluble) and sodium hydroxide. This regenerated sodium hydroxide imparts renewed activity to the absorbent.

Q. Can you predict the exhaustion time of soda lime canister?

Ans. No, we cannot predict the exhaustion time of soda lime canister. This is because the exhaustion time will depend on various factors:

- **Channeling:** The exhaustion time is reduced due to channeling which can result from improper packing or canister design.
- **Size of absorber:** Larger the absorber longer the exhaustion time.

- **Rate of CO₂ production:** Higher CO₂ production, shorter the exhaustion time.
- **Fresh gas flow rate:** High FGF rate longer the exhaustion time.
- The acceptable inspired CO₂ level varies between 0.1 and 1%.
- The position of the absorber in relation to the various components of the circle system will also influence the exhaustion time.

Q. What do you mean by channeling?

Ans. Whenever a gas takes the path of least resistance it is called channeling. If the soda lime canister design is faulty or if the granules are loosely packed, then the gas bypasses bulk of the absorbent and follow the path of least resistance. Channeling usually occurs at the inlet and the inner tube and along the walls. Soda lime near the channels gets exhausted quickly as compared to the other parts. This reduces the efficiency of CO₂ absorption.

However, even after exhaustion there is a blind spot in the distal third of the canister where the granules are still capable of absorbing carbon dioxide.

Q. Why is barium lime not used nowadays?

Ans. Anesthetic agents containing CHF₂ moiety (desflurane, enflurane, and isoflurane) react with warm and dry soda lime and barium lime to produce varying amounts of carbon monoxide. As compared to soda lime barium lime has a greater tendency to produce carbon monoxide, especially if its water content is <2%. The interaction between barium lime and sevoflurane produces very high temperatures. This can cause melting or burning of the components of the absorber. The increase in temperature is much less with soda lime. Due to these problems barium hydroxide is no longer used.

Q. What are the differences between the traditional soda lime and the newer soda lime?

Ans. Traditional soda lime was a high alkali absorbent. It contained high concentrations of potassium and/or sodium hydroxide. The desiccated soda lime would react with volatile anesthetics to produce carbon monoxide. Also sevoflurane is degraded to produce compound A which is nephrotoxic. They do not change color if they are dry. Decrease in moisture content reduces the capacity to absorb CO₂.

The newer absorbents are alkali-free absorbents. They mainly contain calcium hydroxide. Even if they are desiccated there is no carbon monoxide formation with volatile anesthetics. Sevoflurane can be used in a closed circuit with desiccated absorbent as there is little or no compound A formation. They change color even if they are dry and the color does not revert after they are exhausted.

Their CO₂ absorption capacity is not affected when they are dry.

Amsorb is a newer soda lime which contains CaOH, CaCl₂, setting agents (CaSO₄ and polyvinylpyrrolidone). The advantages are lack of sodium and potassium hydroxide eliminates the production of CO and compound A. Absorbs 10.2 L of CO₂ per 100 g of the absorbent.

Lithium hydroxide is also used as a CO₂ absorbent. It reacts with carbon dioxide to form lithium carbonate. It does not react with volatile anesthetics even when it is dry. The major drawback is that it is expensive and can cause burns to the eyes, skin, and respiratory tract if not handled properly. Differences between traditional soda lime and newer soda lime are summarized in **Table 6**.

TABLE 6: Differences between traditional soda lime and newer soda lime.

Component	Soda lime	Baralyme	Medisorb	Dräger-sorb 800+	Amsorb
Ca(OH) ₂ %	84	80	70–80	80	83
NaOH%	2–4		1–2	2	–
KOH%	1–3	May contain some	0.003	2	–
CaCl ₂ % (humectant)					1
CaSO ₄ (hardener)					1
Polyvinylpyrrolidone% (hardener)					1
Water content%	14–19	As water of crystallization	16–20	14	14.5
Ba(OH) ₂ ·8H ₂ O%	–	20	–	–	–
Size (mesh)	4–8	4–8	4–8	4–8	4–8
Indicator	Yes	Yes	Yes	Yes	Yes

Q. What are the problems with the use of inhalational anesthetics with CO₂ absorbents?

Ans. At high temperatures in the soda lime canister, sevoflurane is degraded to fluorinated metabolite called sevo-olefin or compound A. Increasing levels of compound A are seen with increased concentration of agent, prolonged anesthesia, low FGF, increased temperature within the absorber, and desiccated absorbents. Compound A is nephrotoxic in rats however human studies show conflicting results. During closed circuit anesthesia, halothane is degraded to form a halokene which is nephrotoxic in rats but not in humans. Anesthetic agents containing CHF₂ moiety (desflurane, enflurane, and isoflurane) react with warm and dry soda lime and bariumlime to produce varying amounts of carbon monoxide. The production of carbon monoxide increases in the presence of desiccated absorbent,

high temperatures in the absorber, presence of strong alkalis in the absorbent, high FGFs. Carbon monoxide production is greatest with desflurane ≥ enflurane > isoflurane >> halothane = sevoflurane.

Q. What are the advantages of using CO₂ absorber?

Ans. The exhaled gases can be reutilized after CO₂ absorption. It has the following advantages:

- It is economical as low FGFs can be used.
- There is less theater pollution.
- The hazards of explosion are reduced as the inflammable mixtures are enclosed.
- There is conservation of heat and humidity.

Q. If you have a choice between a jumbo CO₂ canister and smaller CO₂ canister; which one would you prefer and why?

Ans. The efficiency of CO₂ absorption depends on:

- How fresh is the soda lime?
- How much surface area of the granules is available for absorption?
- The length of time the gas is in contact with the granules.

The jumbo canisters containing 2 kg of soda lime have a large volume. Due to this more surface area of the granule is available for absorption and the expired gases are in contact with the granules for a longer time. Hence, the jumbo canisters are more efficient in CO₂ absorption. They need to be changed less frequently because they last five times longer than the 500 g canister.

The smaller canisters are easier to maintain, to clean, and have fewer leaks. They are also supplied as disposable cartridges. However, they need frequent changes. Smaller canisters are preferred due to the concerns of carbon monoxide production with dry stale soda lime. Frequent change of the soda lime canister will reduce the risk of carbon monoxide production.

Q. When will you change the soda lime canister?

Ans. The time to change the canister will be indicated by the inspired concentration of CO₂ (when monitoring end-tidal capnometry) and change in the color of the CO₂ absorber.

Q. Can one deliver hypoxic mixture via a circle system?

Ans. When using low FGFs, the exhaled gases exert an increasing influence on the composition of the gases within the circle system. If the inspired concentration of O₂ is gradually falling, it might be due to either of the following reasons:

- *The O₂ supply:* Consumption imbalance. Either the supply is low (reduced O₂ from the flowmeter or reduced alveolar ventilation) or demand is high (hypermetabolic states or high sympathetic activity).
- Denitrogenation process was not complete during the high FGF period (if one is using O₂ and N₂O).

- If nitrous oxide is used, particularly after a long anesthetic, the body gets saturated (equilibrated) with nitrous oxide and its uptake decreases progressively with time, i.e., it accumulates in the circuit. If nitrous oxide is administered at the same flow rate, the fractional concentration of O_2 decreases secondary to reduced uptake of nitrous oxide resulting in a hypoxic mixture. It is mandatory to use inspired oxygen concentration monitor whenever nitrous oxide is used in a closed system.
- Addition of other gases such as helium or desflurane may also affect FiO_2 .

Therefore, it is mandatory to measure O_2 concentration within the circle system, preferably at the patient end.

Q. What are the objectives in a circle system?

Ans.

- Maximum reuse of dead space and fresh gases.
- Maximum venting of alveolar gases (not if the system is completely closed).
- Fresh gas flow should join the inspiratory limb. Some circuits have the FGF conducted via separate tubing (within the inspiratory limb) close to the Y piece. This allows changes in the anesthetic machine to be rapidly reflected in inspired gases. This is important if one uses low flow.
- For pediatric use, low diameter tubes should be used.

Q. What is the ideal arrangement of components in a circle system?

Ans.

- Unidirectional valves should be located between the patient and the reservoir bag one in each limb.
- No gases should flow toward the patient via the expiratory limb during inspiration:
 - Fresh gas flow cannot enter between the patient and expiratory unidirectional valve.
 - Reservoir bag should not be located between the patient and the expiratory valve (**Fig. 14**).

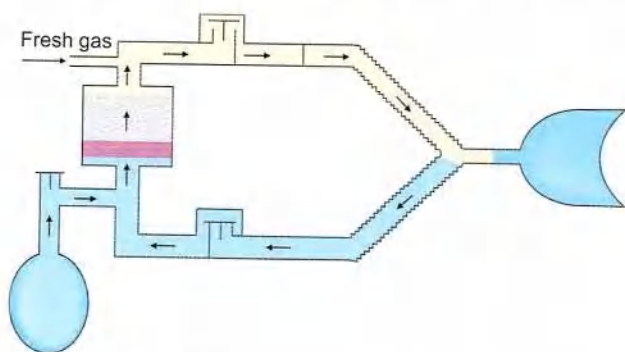


Fig. 14: Functional analysis of circle system.

- No gases should flow from the patient into the inspiratory limb during expiration:
 - APL valve cannot be located between patient and inspiratory valve.
 - Reservoir bag should not be located between the patient and the inspiratory valve.

Certain important considerations:

- The capacity of the reservoir bag should be greater than the patient's inspiratory capacity (~30 mL/kg body weight).
- The soda lime contains 50–70% air around the granules, so for optimal efficiency, the volume of the canister should be at least twice the tidal volume of the patient.

Q. What is low flow anesthesia?

Ans. It is an anesthetic technique via a circle (rebreathing) system in which fraction of recycled (CO_2 free) gases account for at least 50% of the total flow:

- *Low-flow anesthesia* implies the total gas flow rate is <1 L/min.
- *Minimal-flow anesthesia* implies the total gas flow rate is <0.5 L/min.
- *Metabolic-flow anesthesia* implies the total gas flow rate is approximately 0.25 L/min.

Q. What are the advantages and disadvantages of low-flow anesthesia?

Advantages

- *Economical*: Decrease in consumption of gases and inhalational anesthetic agents.
- Effectively warms and humidifies inspired gases.
- *Minimizes atmospheric pollution*: Inhalational agents along with nitrous oxide destroy the ozone layer and thus contribute to the greenhouse effect. Though the contribution to this effect by anesthetist is minimal (every little helps).

Disadvantages

- Initial need for capital investment for anesthesia machine and gas monitoring equipment.
- Increase consumption of soda lime.
- Accumulation of unwanted substances within the circle such as alcohol, carbon monoxide, and methane. Low flow anesthesia is contraindicated in intoxicated patients, and carbon monoxide poisoning.
- Accumulation of products of reaction with soda lime—compound A is formed when sevoflurane is used in the circle system at low flows and high temperature. It has been shown to be nephrotoxic in rats (at a concentration of 250 ppm); however, it is considered safe in humans as the concentrations in the breathing circuit during a typical anesthetic is maximum of 15 ppm. Because of

this concern the US Food and Drug Administration (FDA) set a 2 L/min as the lower limit for carrier gas flow during sevoflurane anesthesia. This was later revised in December 1997 to 1 L/min.

- Technically more difficult compared to conventional anesthetic.

Q. What is the first device that will inform you of a crossover (nonoxygen gas in the oxygen pipeline)? Is it the fail-safe valve or the hypoxic guard?

Ans. It is important to understand that the fail-safe guards against decreased oxygen pressure and not against crossovers or mislabeled contents. As long as there is any pressure in the oxygen line, nitrous oxide (and any other gases) will continue flowing. If oxygen pressure is lost, the fail-safe shuts off the flow of all other gases. The hypoxic guard system works on oxygen pressure as well. It controls the ratio of oxygen and nitrous oxide so that there is minimum 25% oxygen. It does not analyze what is in the oxygen pipeline for the presence of oxygen. *Hence, the oxygen analyzer is the first and only device that will detect a crossover.* If the oxygen analyzer is ignored, then hypoxia will set in. This will be detected by a pulse oximeter on the patient. However, the cause of hypoxia cannot be determined using the pulse oximeter.

Q. For suspected crossover, what two actions must be taken?

Ans.

1. Turn on backup oxygen cylinder.
2. Then disconnect oxygen supply source at the wall.

The pipelines are at a slightly higher pressure (60 psi) than the cylinder regulator (45 psi). If the pipeline supply hose at the wall is not disconnected, the pipeline pressure exerted on the oxygen cylinder regulator diaphragm (downstream side) will prevent the cylinder gas from flowing.

Q. What should you do if you lose oxygen pipeline pressure?

Ans. Just like a crossover:

1. Open the emergency oxygen cylinder fully (not just the three or four quick turns used for checking).
2. Disconnect the pipeline connection at the wall. And always use oxygen analyzer! Alternative ventilation apparatus in the form of AMBU bag with reservoir with flowmeter assembly and tubing should be present in every operation theater to overcome such situations at all times.

Q. The pipeline supply of oxygen has failed. How can you make your emergency E cylinder oxygen supply last as long as possible?

Ans. Oxygen is used as a driving gas for ventilators on the anesthesia machine. If the pipeline supply fails, then using the cylinder gas to drive the ventilator will cause rapid

depletion of the cylinder. In general, the amount of oxygen needed to drive the ventilator is approximately equivalent to the minute ventilation set to be delivered (e.g., TV = 500 mL, RR = 10/min then MV = 5 L). So manually ventilate the patient, assist spontaneous ventilation if possible, use air or nitrous oxide with oxygen if possible, and use low flows.

Q. Your pipeline supply fails and your cylinder gauge shows 800 psi. How long will your emergency oxygen supply last?

Ans. As mentioned earlier the contents of a cylinder containing nonliquefied compressed gases can be estimated from the pressure. According to Boyle's law at constant temperature, the volume of a mass of a gas is inversely proportional to pressure.

Contents in L/Gauge Pressure = Capacity in L/Service Pressure

In the example, X L/800 psi = 660 L/1,900 psi

Therefore, X = 277 L

If oxygen is used at a rate of 2 L/min oxygen, the cylinder will last 138.5 minutes.

Q. What is the safety feature on the back bar?

Ans. The back bar is the horizontal part of the anesthesia machine between the rotameter block and common gas outlet. Vaporizers are mounted on the back bar. They add volatile agents to the fresh gas which is delivered to the common gas outlet. The pressure in the back bar ranges from 7–10 kpa (70–100 cm H₂O) at the rotameter to 1 kpa (10 cm H₂O) at the common gas outlet. The back bar has the selectatec system for mounting the vaporizers. The vaporizers can easily be removed and there is a greater flexibility in the choice and use of vaporizers. The interlocking selectatec system prevents more than one vaporizer being used at the same time. If the locking lever is not engaged, the control dial on the vaporizer cannot be moved. Also the vaporizers can be changed rapidly without interrupting the flow of carrier gas to the patient. There is a pressure relief valve (PRV) which has a threshold pressure of 30–40 kpa. It protects the rotameter block and vaporizers from overpressure damage. There is also a spring loaded nonreturn valve on the back-bar which prevents the back pressure surges from reaching the vaporizers (Datex).

Q. Why do older anesthesia machines have trilene lock on the back bar?

Ans. The trilene vaporizers were always placed downstream of the trilene lock. Whenever trilene was being used this switch diverted all the FGFs directly to the common gas outlet and not to the circle system. This is because CO₂ absorption is an exothermic reaction which produces a lot of heat. At high temperatures trilene gets decomposed to dichloroacetylene and phosgene which are neurotoxic.

Q. What is the preferred bellows design, ascending or descending?

Ans. Bellows are said to be ascending or descending depending on what they do during expiration. An ascending bellows stands, while a descending bellows hangs down during expiration. Hence, it is difficult to detect any disconnections with descending bellows. Since it hangs during expiration it will fill even if it is disconnected from the patient. Also there is a risk of infection due to the collection of exhaled humidity in bellows. As a result of this most modern machines have ascending bellows.

Q. What do you understand by fresh gas decoupling?

Ans. Fresh gas decoupling is a safety feature that has been incorporated into the newer anesthesia workstations that use piston or descending bellows ventilator. In the traditional anesthesia machines which did not have fresh gas decoupling the tidal volume delivered to the patient was a sum of the volume delivered by the ventilator and the fresh gas volume. Depending on the FGF rates there will be an increase in the actual tidal volume that is delivered to the patient. For examples, if the FGF rate is 3 L, set tidal volume is 500 mL, respiratory rate is 10 and the I:E ratio is 1:2. Every minute during inspiration (i.e., 20 seconds) the FGF will be 1,320 mL. 132 mL will get added on to each of the 10 breaths of 500 mL. So the final tidal volume delivered to the patient will be 632 mL. In anesthesia machines with fresh gas decoupling (Fabius GS, Dräger Narkomed 6000), the decoupling valve diverts the FGF into the reservoir bag during the inspiratory phase. This valve is located between the fresh gas source and the ventilator circuit. During the expiratory phase the decoupling valve opens and the gas that has been accumulated in the reservoir bag now gets drawn into the circle system to refill the descending bellows. The excess fresh gas is then vented out through the ventilator exhaust valve that opens during expiration.

Q. What are the advantages and disadvantages of fresh gas decoupling?

Ans. Inappropriate use of the oxygen flush or increasing the fresh gas can result in volutrauma or barotrauma which can be minimized by the use of fresh gas decoupling.

The disadvantage of fresh gas decoupling is that if the reservoir bag is not attached properly or has been removed or if the FGFs are inadequate then there is entrainment of room air which can result in patient awareness or hypoxia.

Q. What is the difference between fresh gas decoupling and fresh gas compensation?

Ans. In fresh gas decoupling, the FGFs are diverted to the reservoir bag during inspiration by a decoupling valve. In fresh gas compensation, the volume and flow sensors give a constant feedback that allows the ventilator to deliver the

set tidal volume regardless of the changes in the FGF rates. Fresh gas compensation is seen in Aestiva and S5 ADU anesthesia workstations.

Q. If there is an emergency life-threatening situation where there is no time to check the anesthesia machine, what is the minimal safety check that must always be done?

Ans.

1. Do a high-pressure test of the breathing circuit which ensures that there is no leak distal to common gas outlet.
 2. During pre-oxygenation observe the movements of the reservoir bag which ensures that there is good mask fit and adequate gas flow.
 3. Check that the suction is working.
- Artificial manual resuscitator should be present in all operation theaters.

Q. Do you know of any intermittent flow anesthesia machines? Where were they used?

Ans. McKessons simpler, Juno Mk II, Walton 5, Cyprane AE, Nargraff: these are few intermittent flow anesthesia machines. They were used for dental anesthesia. They were also called demand flow machines.

Q. How do you calibrate an oxygen analyzer?

Ans. There are two types of oxygen analyzers: a galvanic type sensor (an older "plug in" type), and the paramagnetic. For the galvanic oxygen sensor, first calibrate it to room air. It should take 15–20 seconds to read 21%. If it takes longer than 40–60 seconds, then change the sensor. Then expose to 100% oxygen and ensure it reads close.

Newer paramagnetic sensors use internal calibration routines. So they only need periodic (every 3–6 months) exposure to calibration gas. These sensors are longer lasting.

Q. During a general anesthetic the oxygen analyzer is reading an FiO₂ of 0.2 (and declining) what will you do?

Ans.

- Always trust the monitors until they are proven wrong.
- Call for help.
- Turn on emergency oxygen cylinder and disconnect pipeline from wall.
- If inspired oxygen concentration does not increase in spite of adequate FGF, manually ventilate the lungs with an Ambu bag and room air. Use a portable oxygen cylinder if available.
- Start CPR early.

Q. How would you check for leaks in the low pressure system of the machine?

Ans. Leaks in the low pressure system could be either due to cracks in the rotameter which can result in hypoxic gas mixture or due to leaks in the vaporizers which can lead to

patient awareness. Leaks could also result from failure of tubing connection.

A positive pressure leak test is carried out in machines which do not have outlet check valve (Drager). After shutting off all the gas flows and the vaporizers a known amount of positive pressure (usually about 50 cm H₂O) is applied to the common gas outlet. The manufacturer of each machine would specify how much of leak is acceptable over a specified period of time. This test is again repeated with the vaporizer turned on.

A negative pressure leak test is carried out in machines which have outlet check valves (Datex Ohmeda). All the gas flows and vaporizers are shut off. Then a negative pressure leak test device is connected to the common gas outlet via a 15 mm diameter connector. This device has a squeeze bulb which when evacuated applies a negative pressure of -65 cm H₂O to the low pressure system. The bulb reinflates if there is any leak in the system. This test is repeated with the vaporizer turned on. If it takes >30 seconds for the bulb to reinflate then it is usually acceptable. As the bulb has a volume of 15 mL, reinflation in 30 seconds would indicate a leak of 30 mL/min. The negative pressure leak test is the universal test for all machines.

Q. Describe the procedure for checking a Boyle's anesthetic machine; which uses cylinders as the source of compressed gases.

Ans. If an oxygen analyzer is available, use it. It is the only way to verify the contents of an oxygen cylinder:

- Check that cylinders are securely attached and turned off.
- Open all flowmeter control valves and check there is no flow.
- Turn on oxygen cylinder and check its contents on pressure gauge. Set the rotameter to read 4 L/min. If a second oxygen cylinder is present, turn off the first and check the contents of second. Check there is no flow at the nitrous oxide rotameter. Turn off the second cylinder open the flowmeter and bleed the system.
- Turn on the nitrous oxide cylinder and check the contents on the pressure gauge. Try to start nitrous oxide flow. There should not be any flow of nitrous oxide if the master slave mechanism is functional. If a second nitrous oxide cylinder is present turn it on to check its contents, and then turn it off again.
- Turn off the oxygen cylinder and empty system via oxygen flush. The oxygen warning device should sound (if fitted), and should vent all gases from the machine. There should be no flow at the common gas outlet.
- Turn on the oxygen cylinder again.
- Check that the vaporizers are properly fitted to the back bar, with no leaks. They should contain an adequate amount of volatile anesthetic agent and the controls operate throughout their full range without sticking.

- If the anesthetic machine is fitted with a PRV it should be tested by occluding the common gas outlet with a thumb while gas is flowing. The PRV should open with an audible release of gas. Do not do this test if a PRV is not fitted as it may damage the vaporizers.
- Check your breathing circuit to ensure that it has been assembled correctly, close the valve, fill with gas, and squeeze the reservoir bag to ensure there are no leaks. Open the valves following this check and ensure circuit empties.
- Check the function of other equipment such as Ambu bag, suction apparatus, and laryngoscopes, and ensure that all the drugs, endotracheal tubes, facemasks, and airways you may require are present.

1993 FDA Anesthesia Apparatus Checkout Recommendations are applicable to the majority of anesthesia machines in use worldwide. Since all workstations are not the same, the machine check has to be modified accordingly. 2007 guidelines elaborate only specific systems and subsystems that must be evaluated. Ultimately the user, in conjunction with workstation manufacturers and local policy, should develop institution-specific guidelines for machine check. It is very important to refer to manufacturer's manual for special procedures or precautions related to particular workstations.

Refer to 2008 guidelines for preanesthesia checkout procedures.

<https://www.asahq.org/standards-and-guidelines/2008-asa-recommendations-for-pre-anesthesia-checkout>

Q. How often should a machine check be done and who should do it?

Ans.

Machine check should be done daily.

	To be completed daily	Responsible party
Item 1	Verify that the auxiliary oxygen cylinder and self-inflating manual ventilation device are available and functioning	Provider and technician
Item 2	Verify that patient suction is adequate to clear the airway	Provider and technician
Item 3	Turn on the anesthesia delivery system and confirm that AC power is available	Provider or technician
Item 4	Verify the availability of required monitors, including alarms	Provider or technician
Item 5	Verify that pressure is adequate on the spare oxygen cylinder mounted on the anesthesia machine	Provider and technician
Item 6	Verify that piped gas pressures are > 50 psig	Provider and technician

Contd...

Contd...

	To be completed daily	Responsible party
Item 7	Verify that vaporizers are adequately filled, and if applicable, that filler ports are tightly closed	Provider or technician
Item 8	Verify that there are no leaks in the gas supply lines between the flowmeters and the common gas outlet	Provider or technician
Item 9	Test scavenging system function	Provider or technician
Item 10	Calibrate or verify calibration of the oxygen monitor and check the low-oxygen alarm	Provider or technician
Item 11	Verify that the carbon dioxide absorbent is not exhausted	Provider or technician
Item 12	Check for proper breathing system pressure and leaks	Provider and technician
Item 13	Verify that gas flows properly through the breathing circuit during both inspiration and exhalation	Provider and technician
Item 14	Document completion of checkout procedures	Provider and technician
Item 15	Confirm ventilator settings and evaluate readiness to deliver anesthesia care (ANESTHESIA TIME-OUT)	Provider

	Item to be completed before each procedure	Responsible party
Item 2	Verify that patient suction is adequate to clear the airway	Provider and technician
Item 4	Verify the availability of required monitors, including alarms	Provider or technician
Item 7	Verify that vaporizers are adequately filled and, if applicable, that filler ports are tightly closed	Provider
Item 11	Verify that the carbon dioxide absorbent is not exhausted	Provide or technician
Item 12	Check for proper breathing system pressure and leaks	Provide and technician
Item 13	Verify that gas flows properly through the breathing circuit during both inspiration and exhalation	Provide and technician
Item 14	Document completion of checkout procedures	Provide and technician
Item 15	Confirm ventilator settings and evaluate readiness to deliver anesthesia care (ANESTHESIA TIME-OUT)	Provider

Summarize the safety features on anesthesia machine. Antistatic low friction black rubber wheels to the machine trolley.

• **Pipelines:**

- The wall outlets for pipelines are labeled and color coded for the specific gas.

- Primary valve or automatic shutoff valve at the wall outlet.
- Secondary valve or isolation valve at the wall outlets.
- The Schrader probes or quick connectors for the pipeline terminal outlets are gas specific.
- Also the DISS on the machine end of the pipeline prevents interchangeability of the gas connections.
- The pipeline hoses are color coded.
- **Cylinders:**
 - Color coding
 - Pin index safety system
 - Safety relief valve on the cylinder
 - Symbol of the gas on the cylinder valve
 - Label on the cylinder
 - Filling on cylinder within the service pressure.
- Check valves at the cylinder inlet and pipeline inlet on the machine.
- Bourdons pressure gauge.
- Pressure regulators.
- Pressure relief valves downstream of the pressure regulators.
- Oxygen flush valve for emergency oxygen.
- Oxygen failure safety alarm.
- Oxygen supply pressure failure safety device.
- Hypoxia guard at the flowmeter.
- **Flowmeters:**
 - Color, touch and placement coding
 - Rotating bobbin
 - Antistatic spray in the flowmeters
 - Downstream placement of oxygen flowmeter.
 - Radio-fluorescent plastic sheet behind flowmeters.
- The interlocking selectatec system prevents more than one vaporizer being used at the same time. If the locking lever is not engaged the control dial on the vaporizer cannot be moved. The selectatec block on the back bar of the anesthesia machine allows the vaporizers to be changed rapidly without interrupting the flow of carrier gas to the patient.
- **In the older machines:**
 - Placement of the vaporizer
 - Copper plunger in Boyle's bottle
 - Trilene interlock with closed circuit (in older machines)
 - Black tubing's of breathing systems.
- Nonreturn valve downstream of the vaporizer prevents back pressure which might cause output of high concentration of vapor (Datex).
- Pressure relief valve situated downstream of the vaporizer prevents damage to the flowmeters or vaporizers if the common gas outlet is obstructed.
- Soda lime and its indicator in closed circuit.

■ GAS LAWS

Boyle's Law

For a constant amount of gas at a constant temperature, the product of the pressure and volume of the gas is a constant:

$$P_1V_1 = P_2V_2$$

Charles's Law

For a constant amount of gas at a constant pressure, the volume of the gas is directly proportional to the absolute temperature:

$$V_1/T_1 = V_2/T_2$$

Gay-Lussac's Law

At a given temperature and pressure, equal volumes of gas contain equal numbers of moles. This is sometimes also known as Avogadro's law:

$$P_1/T_1 = P_2/T_2$$

Avogadro's Hypothesis

Equal volumes of gases at the same temperature and pressure contain equal numbers of molecules.

Thus, 1 mole of any gas (i.e., 6.02×10^{23} gas molecules) at 1 atmosphere pressure and 0°C occupies a volume of approximately 22.4 L.

Each of these laws is a special case of a more general law. That general law is called the ideal gas law:

$$PV = nRT$$

At standard temperature and pressure (commonly abbreviated as STP), the value of the temperature is 0°C

(273 K) and the pressure is at 1 atmosphere 760 mm Hg or 760 torrs or $1.01 \times 10^5 \text{ Nm}^{-2}$ (Pascals).

This implies that at STP, 32.0 g of oxygen gas will occupy a volume of 22.41 L. In addition, 44.0 g of nitrous dioxide will also occupy a volume of 22.41 L at STP.

Dalton's Law

Dalton's law of partial pressure states that the pressure of a gas mixture is the sum of the partial pressures of the individual components of the gas mixture.

Graham's Law of Diffusion

The rate at which gases diffuse is inversely proportional to the square root of their densities.

Henry's Law

Henry's law states that at a given temperature, the amount of gas dissolved in a solute is directly proportional to the pressure of the gas above the substance.

■ SUGGESTED READING

1. Davey J, Ali D. Ward's Anesthetic Equipment, 5th edition. Philadelphia, PA: Saunders/Elsevier; 2005.
2. Dorsch JA, Dorsch SE. Understanding Anesthesia Equipment, 5th edition. Philadelphia, PA: Wolter Kluwers/Lippincott Williams and Wilkins; 2008.
3. Miller RD. Miller's Anesthesia, 6th edition. Philadelphia, Elsevier, Churchill, Livingstone; 2005
4. Parbrook GD, Davis PD, Parbrook EO. Basic Physics and Measurement in Anesthesia, 4th edition. Oxford, United Kingdom: Butterworth-Heinemann; 1995.

Vaporizers

Priya Ranganathan, Madhavi Shetmahajan

Q. Define the following and explain the importance to anesthesia.

Ans.

Critical temperature: The temperature above which a substance in the gaseous phase cannot be condensed into a liquid, no matter how much pressure is applied.

Vapor and gas: A vapor is a substance that is in the gaseous phase below its critical temperature. This means that the vapor can be condensed to a liquid or to a solid by increasing its pressure without reducing the temperature, e.g., water vapor is the vapor form of water. The term "gas" is used to describe a substance that is in the gaseous phase above its critical temperature, e.g., nitrogen is a gas at room temperature. It is important to know the difference between gases and vapor because gas laws are applicable to gases and may not always be applicable to vapors at normal room temperature. Oxygen and nitrogen are gases whereas carbon dioxide, halothane, and ether are vapors. The critical temperature of nitrous oxide is 36.5°C. So, at room temperature of 20°C, it is a vapor. However, in the tropics where the atmospheric temperature is >36.5°C, it is a gas.

Vaporizer: A vaporizer is an instrument designed to facilitate the change of a liquid anesthetic into its vapor and add a controlled amount of this vapor to the flow of gases going to the patient.

Vapor pressure: It is the pressure exerted by the molecules escaping from the surface of a liquid to the gaseous phase in a closed container.

Saturated vapor pressure: When the liquid and its vapor are in equilibrium, the vapor pressure is now called the saturated vapor pressure (SVP). SVP is dependent on only the temperature and nature of liquid.

Vapor pressure of an agent determines how much of vapor will be formed from 1 mL of the liquid. Since different anesthetic agents have different vapor pressures,

they need separate vaporizers, and the vaporizer cannot be interchanged between agents.

Boiling point: The temperature at which SVP becomes equal to atmospheric pressure and at which all the liquid agent changes to the vapor phase is the liquid's boiling point.

Vaporizer output: Refers to the concentration of vapor at the outlet of a vaporizer.

Vaporizer concentration: Denotes the concentration delivered by a vaporizer when fresh gas containing no vapor passes through it.

Therefore, in an out-of-system vaporizer, vaporizer output equals vaporizer concentration. In an in-system vaporizer, expired gases contain some volatile anesthetic, and therefore vaporizer output can exceed the vaporizer concentration.

Vaporizer capability: Refers to the maximum concentration that can be delivered by a vaporizer at the highest setting of the concentration dial. Manufacturers decide the vaporizer capability based on the minimum alveolar concentration (MAC) of the agent. For example, sevoflurane has a higher MAC than isoflurane and therefore needs a vaporizer with a higher capability (maximum 8% in case of sevoflurane as compared to 5% with isoflurane).

Vaporizer efficiency: Means the ability of a vaporizer to saturate the carrier gas passing into the vaporizing chamber. Vaporizer efficiency is increased by using wicks, baffles, and longer vaporizing chambers to increase the surface area available for vaporization.

Concentration calibrated vaporizers are constructed with the assumption that gas in the vaporizing chamber is saturated, i.e., SVP of volatile agent is achieved. Inability of vaporizer to achieve SVP will result in inaccuracy of vaporizer.

Q. How are gas concentrations expressed?

Ans. Two methods are commonly used to express the concentration of gas or vapor: (1) partial pressure and (2) volumes percent.

1. *Partial pressure:* The part of the total pressure due to any one gas in the mixture is called the partial pressure of that gas.
2. *Volumes percent:* It is defined as the number of units of volume of a gas in relationship to a total of 100 units of volume for the total gas mixture.

$$\frac{\text{Partial pressure}}{\text{Total pressure}} = \frac{\text{Volumes percent}}{100}$$

Q. Why is ethyl chloride kept under pressure?

Ans. Ethyl chloride has a boiling point of 12.5°C and is therefore a gas at room temperature. In order to liquefy it, it needs to be pressurized.

Q. Calculate the amount of vapor produced by 1 g of its liquid agent. How much liquid agent does a vaporizer use per hour?

Ans. 1 mole of a gas weighs 1 g molecular weight. The molecular weight of isoflurane is 185.

Therefore, 1 mole of isoflurane vapor weighs 185 g.

As per Avogadro's hypothesis, 1 mole of a substance = 22.4 L of gas

22.4 L of isoflurane vapor weighs 185 g

Therefore, density of isoflurane vapor is $185/22.4 = 8.25$ g/L. Therefore, 1 g = $1,000/8.25$ mL of vapor

Density of isoflurane liquid is 1.5 g/mL. Therefore, 1 g = $1/1.5$ mL of liquid

$1/1.5$ mL of liquid = $1,000/8.25$ mL of vapor

1 mL of liquid = $1,000/8.25 \times 1.5 = 180$ mL vapor

For most modern agents, 1 mL of liquid volatile agent yields about 200 mL vapor.

The amount of vapor used per minute = fresh gas flow rate $\times$ time $\times$ concentration setting

For example, to deliver 1% isoflurane at FGF of 2 L/min for 60 minutes, one would need

$1/100 \times 2,000 \text{ mL} \times 60 \text{ min} = 1,200 \text{ mL}$ of isoflurane vapor.

This would correspond to $1,200/180 = 6.7 \text{ mL}$ of liquid isoflurane

Another formula which can be used with most agents is $3 \times \text{Fresh gas flow (FGF) (L/min)} \times \text{volume \%} = \text{mL liquid used per hour}$.

Q. Classify vaporizers with examples.

Ans. The most commonly used classification is the Dorsch and Dorsch classification.

- *Method of regulating output:*
 - Variable bypass: Ether bottle, TEC vaporizers
 - Measured flow: Copper kettle, side-arm vernitrol.

- *Method of vaporization:*

- Flow over:
 - With wick—TEC vaporizers
 - Without wick—Goldman bottle
- Bubble through: Copper kettle
- Flow-over or bubble-through: Ether bottle, depending on the position of the plunger
- Injection: Desflurane, Kion though desflurane vaporizer has been described as an "injector", it is more ideally classified as a gas-vapor blender.

- *Temperature compensation:*

- Thermocompensated:
 - By altered flow—TEC vaporizers
 - By supplied heat—copper kettle
 - By both supplied heat and altered flow: EMO (Epstein-Macintosh-Oxford).
- Non-compensated: Ether bottle.

- *Specificity:*

- Agent specific: TEC vaporizers
- Multi-agent: Goldman bottle.

- *Resistance:*

- Plenum (high resistance): TEC vaporizers
- Draw over (low resistance): Goldman bottle, EMO.

- *Location:*

- In circuit: Vaporizer in circle (VIC)—EMO, Goldman bottle
- Out of circuit: Vaporizer outside circuit (VOC)—TEC vaporizers.

Vaporizers may or may not be concentration calibrated. For example, the Goldman vaporizer is a variable-bypass vaporizer. However, the calibrations are arbitrary and do not indicate actual concentrations delivered. The desflurane vaporizer has concentration calibrations. However, it is neither measured-flow nor variable-bypass.

Q. Describe the functioning of variable bypass vaporizers with an example.

Ans. Variable bypass vaporizers accept the total gas flow from the anesthesia machine and deliver the gas flow with a predictable concentration of vapor to the common gas outlet (**Fig. 1**).

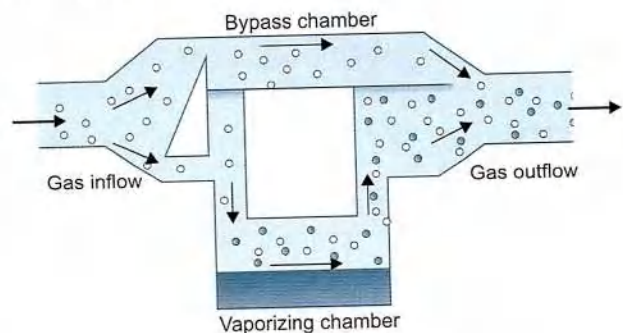


Fig. 1: Variable bypass vaporizer.

The carrier gas flow entering the vaporizer is divided into two parts—one part going through the bypass chamber and the other part going through the vaporizing chamber where it is saturated with the vapor of the liquid anesthetic agent.

Q. Define splitting ratio.

Ans. The ratio of the carrier gas flows through bypass and vaporizing chambers is called the splitting ratio:

- SVP of halothane at 20°C is 243 mm Hg.
- At 20°C and 760 mm Hg, halothane in the vaporizing chamber will contribute to $243/760 = 32\%$ of volume (as per Dalton's law of partial pressures).
- Remaining 68% is made up of carrier gas.

Therefore, if 100 mL of carrier gas enters, it picks up $100/68 \times 32 = 47$ mL of halothane:

- Therefore, 147 mL of carrier gas + vapor exits the vaporizer chamber.
- We want 1% halothane at total gas flow of 5 L/min to patient.
- We want 50 mL of halothane.
- Remaining 4,950 is carrier gas.
- We know that 100 mL gas picks up 47 mL halothane vapor.
- To pick up 50 mL halothane, we need 106 mL gas.
- So 106 mL gas enters the vaporizing chamber and rest 4,844 mL gas (4950–106) goes through the bypass.
- Therefore, for 1% halothane the splitting ratio is 106:4,844 or 1:46 at 20°C.

The splitting ratio is affected by the concentration setting on the dial, and by the temperature-compensating mechanism in the vaporizer.

When the temperature of the volatile agent changes, the SVP in the vaporizing chamber changes. If the splitting ratio is kept the same, this will result in change in the vaporizer output. Therefore, temperature compensating mechanism are incorporated which change the splitting ratio by altering flow through the vaporizing chamber or the bypass chamber, e.g., when the temperature of the volatile agent drops, the carrier gas passing through the vaporizing chamber will pick up less agent (it has lower vapor pressure and therefore less volume percent) and thus lead to drop in vaporizer output. This is compensated by increasing the proportion of flow going into the vaporizing chamber so that the vaporizer output is maintained.

Q. Splitting ratios for different agents—can vaporizers be interchanged?

Ans. Different anesthetic agents have different SVPs at a given temperature. Therefore, the partial pressure exerted by the vapor in a mixture of gases will be different. Hence, splitting ratios are agent-specific and vaporizers cannot be interchanged unless two vapors have the same vapor pressure.

Q. Describe the functioning of a measured-flow vaporizer.

Ans. This type of vaporizer utilizes a measured flow of carrier gas, usually oxygen, to pick up anesthetic vapor (**Fig. 2**). The vaporizer system consists of three parts:

1. A vaporizer fitted with a thermometer
2. A flowmeter assembly
3. A vaporizer circuit control valve.

A measured amount of carrier gas flows through a flowmeter and onto the vaporizer circuit control valve, which directs it to the vaporizer. The gas saturated with vapor is returned to the vaporizer circuit control valve, where it is diluted by the remaining fresh gas flow and goes to the patient.

To calculate the vaporizer output, one must know the vapor pressure of the agent, the atmospheric pressure, the total flow of gases and the flow to the vaporizer.

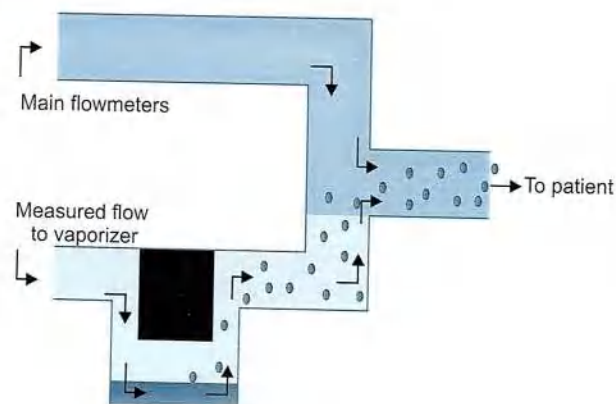


Fig. 2: Measured-flow vaporizer.

$$\% \text{ concentration of agent} = 100 \times \frac{\text{vaporizer output of anesthetic}}{\text{total flow}}$$

It is important to remember that:

- The oxygen flowing through the vaporizer should be counted as additional to that going directly to the patient.
- The vaporizer should not be turned on until the flows on the other flowmeters have been set—otherwise a lethal concentration of agent may be delivered to the patient.

Q. What does method of vaporization mean?

Ans. The method of vaporization refers to the pathway followed by the carrier gas as it travels through the vaporizing chamber.

Q. What are the factors affecting efficiency of vaporization in a flow-over vaporizer?

Ans.

- *Area of the gas-liquid interface:* This can be increased by using wicks or baffles.

- *Velocity of carrier gas flow:* Time is required to establish equilibrium between the fluid and its vapor, and this may not be possible at very high gas flows. Also at very low flows, the carrier may not be able to pick up the vapor leading to a lower efficiency.
- *Height of the gas flow above the liquid:* Gases passing closer to the surface of the liquid agent pick up more vapor than gases flowing at a height over the liquid. For example, in the ether bottle, vaporization can be increased by depressing the plunger so that gases pass under the hood and over the surface of the liquid.

Q. What are the factors affecting efficiency of vaporization in a bubble-through vaporizer?

Ans.

- *Size of the bubbles:* The smaller the bubbles, the more are the surface area available for equilibration. For example, in the copper kettle, a sintered bronze plate is used to break the gas flow into small bubbles to increase efficiency of vaporization.
- *Depth of the liquid:* The deeper the liquid, the more is the time required for bubbles to reach the surface, allowing more time for equilibration. For example, in the ether bottle, depressing the plunger below the level of the liquid increases vaporization.
- *Velocity of carrier gas flow:* The faster the flow of bubbles, the less is the time available for equilibration.

Q. Define heat of vaporization, specific heat, thermal capacity, and thermal conductivity.

Ans.

- *Heat of vaporization:* It is the amount of heat required to vaporize 1 g of a liquid at its boiling point with no change in temperature. Heat required to vaporize anesthetic agents is drawn from the remaining liquid and the surroundings. As vapor is generated, the temperatures of the vaporizer and remaining liquid fall. This causes the vapor pressure to decrease and would result in decreased vaporizer output if no compensatory mechanism were provided.
- *Specific heat:* The specific heat of a substance is defined as the quantity of heat required to raise the temperature of 1 g of the substance through 1°C. Specific heat is important when considering the amount of heat that must be supplied to an anesthetic agent to maintain a stable temperature when heat is lost through vaporization. Specific heat is also important in the choice of material from which a vaporizer is constructed. A substance with a high specific heat (and high thermal capacity) will change temperature more slowly, and will therefore provide a more stable temperature for vaporization thereby delivering stable vaporizer output.

- *Thermal capacity:* Product of specific heat and mass (amount of heat stored in the vaporizer body). Therefore, vaporizers are heavy due to large amount of material used in its construction.
- *Thermal conductivity:* It is a measure of speed with which heat flows through a substance. Substances with a high thermal conductivity can equilibrate the temperature of its contents with the atmosphere, thereby providing a more stable temperature.

Copper has a moderately high specific heat (0.1 cal/°C/g) and a very high thermal conductivity. Hence, it is used in the construction of a number of vaporizers like the copper kettle. The copper kettle is placed on a copper surface to further enhance conductivity.

Q. What are the different techniques of temperature compensation in vaporizers?

Ans. Temperature compensation in vaporizers can be by:

- *Supplied heat:* Electrically heated—TEC 6 vaporizer for desflurane.
- *Altered flow:* At lower temperatures, a higher percentage of carrier gas flows through the vaporizing chamber to compensate for the drop in the SVP, e.g., bimetallic strip—TEC vaporizers, bellows valve—EMO, annular valve of dissimilar metals in some types of concentration calibrated modern vaporizers.

Temperature buffered:

- Water jacket in the EMO helps maintain stability of temperature due to its high thermal capacity.
- Copper kettle kept on copper surface helps to reduce temperature fluctuations by conducting heat from atmosphere to the volatile agent.

Q. What are the differences between in-system and out-of-system vaporizers?

Ans. Vaporizers are classified as being in or out-of-system depending on their location in relation to the breathing circuit (**Table 1**).

Out-of-system vaporizers:

All measured flow vaporizers are located out of the breathing system and have a dedicated flowmeter. Plenum vaporizers (high-resistance) are also used out-of-system. These may be attached:

- Between the flowmeters and the common gas outlet:
 - Advantage is that the vaporizer is securely mounted on the machine. Modern machines have their vaporizers sited in this location.
- Between the common gas outlet and the breathing system (when Mapleson systems are used):
 - Advantage is that it is more convenient to move the vaporizer from one location to another

- Disadvantages are:
 - Chances of tipping
 - Can have incorrect connections (reversal of flow through vaporizer)
 - Flush may cause agitation of liquid agent and excessive delivery but back pressure is not possible if Mapleson system used. In circle system, this location is not possible.

TABLE 1: Differences between in-system and out-of-system vaporizers.

In-system vaporizers	Out-of-system vaporizers
Low resistance is required as patient breathes through vaporizer. Drawover vaporizers and vaporizers in circuit are in-system vaporizers, e.g., Goldman, EMO (Epstein-Macintosh-Oxford)	Low and high resistance vaporizers can be used as resistance is overcome by pressure of carrier gas, e.g., TEC 3 onward
Expired gas passes through vaporizer increasing vaporizer output. Inspired anesthetic concentration (IAC) is always higher than the dialed concentration. Increasing gas flows decrease IAC to approximate dialed concentration	Vaporizer output and vaporizer concentration are equal. Inspired anesthetic concentration is lower than the dialed concentration
Vaporizer with low capability desirable	Vaporizers must have high capability
Vaporizer output is dependent on minute ventilation	Vaporizer output is not dependent on minute ventilation
Condensation of water vapor from the expired gases into the volatile liquid impairs vaporization of the volatile liquid	No risk of condensation as expired gases do not pass through the vaporizer
Must possess easy access for washing and drying because it is exposed to expired gases from the patient	It does not require cleaning and washing

In-system vaporizers: In-system vaporizers are those in which the patient's inspiratory and expiratory gases go through the vaporizer. The typical use of in-system vaporizer was:

- **As inhalers:** Air (with/without supplementing oxygen) is drawn in-and-out through the vaporizer by the patient's spontaneous respiration.
- Similar circuit as inhaler but with equipment for assisting ventilation.
- **In the circle system:** Either in the inspiratory or the expiratory limb.

When vaporizer is in expiratory limb, there is higher risk of condensation of water vapor from the expired gases into the volatile liquid which can impair vaporization of the volatile liquid.

With in-system vaporizers, vaporizer output is unpredictable and could be very high because the expired

gas passing through the bypass chamber also contains vapor. Historically, low resistance vaporizers with poor efficiency, e.g., Goldman's vaporizers were used as in-system vaporizer to improve vaporizer output with low risk of very high vapor concentration (other method used to improve vaporizer output with Goldman vaporizer with Mapleson systems was to connect two Goldman vaporizers in series). Potent volatile agents and efficient vaporizers are not advisable for in-system use.

Plenum vaporizers cannot be used as in-system vaporizers mainly because they offer resistance to breathing, both during inspiration and expiration making them undesirable both during spontaneous respiration and intermittent positive pressure ventilation (IPPV). Also, if used in the inspiratory limb of the circle-system during IPPV, the vaporizer receives very high flow rates where it may not perform efficiently.

Q. Describe the back pressure changes that occur in vaporizers with the use of positive-pressure ventilation.

Ans. The use of controlled or assisted ventilation leads to transmission of positive pressure along the breathing system to the vaporizer. This can also occur with the use of the oxygen flush device. The intermittent back pressure can lead to:

- **Pumping effect:** Increase in vaporizer output
- **Pressurizing effect:** Decrease in vaporizer output.

Mechanism of Pumping Effect

In variable bypass vaporizers, an increase in positive pressure at the outlet causes compression of gas in the vaporizing chamber and bypass. When the back pressure decreases suddenly, the pressure in the bypass chamber falls more quickly because it is a smaller chamber, with less resistance to flow. The gas in the vaporizing chamber which is saturated with vapor exits from both the outlet and the inlet of the vaporizing chamber, and thus vapor-rich gas enters the bypass. Therefore, the overall vaporizer output is increased.

In measured-flow vaporizers, when back pressure is applied, gas is forced back into the vaporizing chamber where it picks up additional vapor, thus increasing vaporizer output.

The pumping effect is more with low flows, low dial settings, and when the level of liquid agent in the vaporizer is low:

Modifications to Minimize Pumping Effect

Modifications in variable-bypass vaporizers:

- **Changing the size of the bypass and the vaporizing chamber:**
 - By making the vaporizing chamber smaller and the bypass larger—this TEC 4 vaporizer has a much smaller vaporizing chamber than the TEC 2.

- *Preventing spillage from the vaporizing chamber into the bypass chamber:*
 - By making the inlet of the vaporizing chamber longer
 - By excluding wicks at the inlet of the vaporizing chamber
 - By incorporating a baffle system or a positive pressure assembly in the vaporizing chamber.
- *Back pressure valves:*
 - A one-way valve can be inserted at the vaporizer outlet.

Modifications in measured-flow vaporizers:

- Smaller vaporizing chamber
- Longer outlet tube
- Relief valve or check valve at the outlet.

Modifications in the machine:

- Pressure relief valve or check valve to limit transmission of pressure to the vaporizer.
- As per ASTM machine standard, the pressure transmitted to the vaporizer during use of oxygen flush should not be >100 cmH₂O above the normal working pressure, and the vaporizer output should not vary by $>20\%$ when the oxygen flush is used.

Mechanism of Pressurizing Effect

Pressurizing effect occurs when carrier gas is compressed in the vaporizing chamber. However, since the number of molecules of vapor in the vaporizing chamber does not increase, the carrier gas cannot pick up additional molecules of vapor. This results in a dilution of anesthetic in the vaporizing chamber and a decrease in vaporizer output.

The pressurizing effect is more at high gas flows. There is usually an interplay between the pressurizing and pumping effects. Changes in vaporizer output are more with the pumping effect and are of greater clinical significance.

Q. What are the hazards of vaporizers?

Ans.

- *Incorrect concentration delivered:*
 - High concentration:
 - Liquid anesthetic in delivery tube
 - Pumping effect
 - Reversed flow
 - Agitation
 - Low concentration:
 - Cooling of liquid
 - Pressurizing effect
 - Wicks covered with liquid
 - Very low/high flows.
- *Incorrect agent:* Use of the wrong agent in the vaporizer can lead to delivery of dangerously high concentrations. This is minimized by:
 - Agent-specific filling devices

- Color coding
- Use of agent monitors.
- *Tipping:* This can lead to entry of liquid agent into the bypass chamber. Dangerously high concentrations of vapor will be delivered when the vaporizer is turned on. In free-standing vaporizers, liquid agent can directly enter the breathing circuit and the patient's lungs. This can be prevented by:
 - Mounting vaporizers on the manifold
 - Draining the vaporizer before being moved
 - Newer vaporizers like the TEC 4 are designed to avoid spillage (baffle system) even when turned 180°
 - The TEC 6 vaporizer for desflurane goes into standby mode if it is tilted $>10^\circ$. If tipping is suspected, the vaporizer should be flushed with high gas flows for 15–20 minutes before being used.
- *Overfilling:* The problems with overfilling are:
 - Liquid agent can enter the fresh gas line leading to high concentrations
 - If the wicks are completely submerged, the surface area for vaporization is decreased, leading to a fall in vaporizer output. This is prevented by:
 - Low level filling port and liquid level indicator glass
 - Use of agent specific filling devices
 - Filling only up to level indicated.
- *Reversal of flow:* With reversal of flow through the vaporizer, output is increased. This can be prevented by:
 - Mounting the vaporizer on the manifold
 - In free-standing vaporizers, use of the arrows to indicate the direction of gas flow.
- *Leaks:* Can lead to:
 - Wastage of agent
 - Delivery of wrong concentrations
 - OT pollution
 - Prevented by:
 - Machine check including negative pressure check
 - Vigilance for vaporizers needing excessive filling or for odor of agent
 - Agent monitoring.

Q. Enumerate the safety features of vaporizers.

Ans.

- *Agent-specific filling systems:*
 - Prevent accidental filling with wrong agent
 - Reduce air pollution during filling
 - Prevent water and contaminants from entering the vaporizing chamber.
- Consist of a color-coded bottle collar and a matching color-coded adaptor, which fits into the vaporizer filling receptacle.

- *Low filling port:* This minimizes overfilling because the filler port is located at the maximum safe liquid level.
- Secured vaporizers (less ability to move them about minimizes tipping).
- *Interlocks:* Prevent administration of more than one inhaled anesthetic at a time.

Concentration dial increases output in all when rotated counterclockwise (as seen from above).

Q. Classify and give a brief description of Flagg's can.

Ans. Flagg's can consist of a square tin can, later replaced by plastic. The lid had a hole through which a rubber tube was used to connect the can to the endotracheal tube. Side holes in the lid allowed entry of air and could be partially closed to adjust the concentration of ether. The area of the holes was equal to the area of the trachea, to minimize resistance to breathing. Oxygen could be supplemented by inserting a catheter connected to oxygen source via one of the holes. The tip of this catheter should be kept above the level of the liquid ether to prevent bubbling of ether leading to dangerously high concentrations. It functions as an inhaler (in-system vaporizer). The KEM bottle is a modification of Flagg's can.

Hazards of Flagg's Can

Tipping, leading to direct entry of ether into the tube and respiratory tract. If side holes were completely obstructed, hypoxic mixture will be delivered to the patient.

Q. Describe devices used for ether anesthesia.

Ans.

Schimmelbusch Mask and Yankauer's Mask

Schimmelbusch mask and Yankauer's mask are used for open-drop method of anesthesia. They could be used with ether, chloroform, ethyl chloride, etc.

Metal frame over which gauze or lint is spread. In case of ether, the liquid is dropped evenly over the whole surface, whereas with chloroform, it is restricted to one-half of it to ensure that air is freely drawn and the concentration of the vapor is not too high.

For ether, 16 layers of gauze are used whereas for chloroform or ethyl chloride, 12 layers of gauze or (295.7 mL) 1 layer of lint are applied.

When ether is being used, the high rate of vaporization requires so much latent heat that the mask becomes cool and water vapor from the exhaled air may condense on it and freeze. This causes resistance to flow of air through the gauze and also reduces the rate of vaporization. Therefore, a second mask or a fresh supply of gauze is needed during long administration.

When using the Schimmelbusch mask, the patient's face is covered with a piece of gamgee with a central hole

to expose the patient's nose and mouth. This prevents vapor or liquid from entering the patient's eyes and also reduces the amount of air drawn in by the patient, between the mask and his face. A piece of gamgee may also be used to cover the mask.

Ogston's Inhaler

Semi-open drop anesthesia. Consists of a mask surmounted by a wire frame around which a napkin or some gauze could be erected to keep the vapor trapped.

Q. Classify and give a brief description of Boyle's bottle.
Ans.

Boyle's Bottle (Vaporizer)

Classification

- Variable bypass, flow over without wick or bubble through, out of system
- No temperature compensation, not calibrated
- *Agents used are:*
 - Ether
 - Methoxyflurane (MOF)
 - Trilene
 - Chloroform
 - Halothane.

Function

With the lever in "off" position—gas flows through bypass entirely. As the lever is turned toward the "on" position, an increasing proportion of gases pass through the bottle. In full on position, all gases pass through bottle. In Boyle's bottle, the vaporizing chamber is much larger than the bypass chamber unlike modern vaporizers.

When the cowl is up, gases flow directly from the U-tube to the outlet of the vaporizer.

Gas may be diverted by lowering the cowl over the end of U-tube and gases are forced to impinge on surface of ether. If cowl is fully down, then gas bubbles up through liquid to produce maximum vaporization. The position of the cowl is adjusted by a plunger, which passes through a gland to maintain a gas-tight seal.

The control drum rotates inside the body of the vaporizer and is positioned laterally by adjusting and locking rings at each end. Special grease is used to maintain a seal and yet allow rotation. There is spare grease in the grease injector cap (at the back of the vaporizer), and by turning this, it can be fed into the seating of the drum. As gases and vapors pass through the vaporizer, the grease tends to be slowly washed away and this results in stiffness if extra grease is not added.

The plunger may become loose and tend to fall down on it. This can be corrected by tightening the gland nut. The packing in the gland may be of cotton, neoprene and nylon, and needs replacement when it wears out.

There is a filling orifice that is blocked by a cork stopper, which is retained by a small chain. The cork should have a good fit to prevent leak of gases. In some vaporizers, the metal anchor for the retaining chain passes through the cork. If the chain is broken, the metal core of the cork can act as a spark plug if someone who is charged with static electricity touches it. Hence, it is important that the chain is intact. Sometimes, the top of the cork is insulated to prevent this hazard.

The bottle sealing washer is made of cork and may have a canvas or metal insert. If this is damaged, there may be a leak of anesthetic gases and vapor. Copper in ether bottle acts as an anticatalyst preventing decomposition of ether. Some bottles are colored (brown/green) to prevent decomposition of volatile agent by light, e.g., trilene.

Two bottles are available—broad one for ether and a narrow one for other agents. High concentrations (15–20%) of ether are required in the early phases of inhalational anesthesia due to its high blood gas solubility and therefore a larger surface area and larger capacity bottle is required.

Evaluation and Use

Graduation of control lever is arbitrary and as a rule it starts functioning at mark II. The concentration of vapor also depends on the temperature, the level of liquid anesthetic agent and the rate of gas flow. Temperature is maintained by wiping bottle from outside or by wrapping the bottle with warm gamgee.

Ether vaporizer—knob is turned on slowly, usually one-quarter of a division after four consecutive regular breaths. Ether bottle—270 mL of ether is used. Trilene and chloroform— $\frac{1}{2}$ inch in depth of anesthetic used. Volatile agents used in the vaporizer must be removed from the bottle after use and be discarded in order to prevent a risk of being used in other bottle.

Minimum ether concentration with 10 oz ether filled with 8 L gas flow is 4.5%. Maximum concentration with cowl at highest position is 14.5%. Maximum concentration with cowl fully depressed for maximal bubbling is 40%, but this falls rapidly as the ether cools and temperature falls.

In case of chloroform and trichloroethylene, a sufficient concentration is produced by turning the control knob fully on and depressing the plunger may lead to dangerously high concentrations.

This bottle may not produce adequately high concentrations for induction with MOF.

Cleaning and Care

- Liquid is removed after use.
- Clean and dry vaporizer.

Hazards

- Lever gets stuck due to thymol, which is used as a preservative.

- Plunger may get stuck.
- Ether is very volatile and there may be a build-up of vapor in the bottle when it is turned off, especially in warm environments. This can lead to a surge of high-concentration ether vapor when the vaporizer is turned on. This can be prevented by running a small quantity of gases through the vaporizer prior to commencement of anesthesia, or by removing the cork from the filler orifice for a brief period. Since agents are not color coded, the residual agent in the Boyle's bottle may be poured back into the wrong bottle. It is preferable to discard residual agent, to avoid this mishap.

Q. What are the differences between the Boyle's bottles for ether and trilene?

Ans. The ether bottle has a broad base to allow greater surface area for evaporation. It has markings up to 300 cc. The trilene bottle is narrow to prevent excessively high concentrations of vapor. It has markings up to 100 cc.

Q. What is the trilene interlock?

Ans. Trilene combines with soda lime to form phosgene which is a toxic gas. The trilene interlock is present on the back bar of some machines which does not allow the operator to switch on the trilene vaporizer when machine is on circle-system and vice versa. This ensures that trilene cannot flow to the soda lime canister.

Q. Classify and describe EMO.

Ans.

- Concentration calibrated, variable bypass
- Flow over with wicks
- Draw over (low internal resistance)
- Agent specific
- Temperature compensated by supplied heat and by altered flow
- Vaporizer in and out of system
- The EMO system consists of vaporizers alone or in series, valves, bellows or self-inflating bags, and accessories.

EMO Inhaler (Epstein and Macintosh, 1956)

Construction

It is 23 cm in diameter, 24 cm in height and weighs 6.5 kg with the water compartment full. It has an annular ether vaporizing chamber (V), lined with wicks. Surrounding this is a water compartment (W) and an automatic thermocompensating valve (T). The air passages are relatively large, offering a low resistance to breathing and very low resistance to flow (<1.25 cmH₂O at 40 L/min).

The air inlet communicates with a large chamber with two outlets. One gives access to the main ether chamber via the closing mechanism (L) which seals off the entry to the ether chamber when the control indicator is in the "transit" position. Also incorporated in the closing mechanism is

an inlet relief valve, which allows air to enter the inhaler if the main air inlet becomes blocked. The other outlet port leads into the small chamber in the center of the control rotor which is fully open in the "transit" position but as the concentration indicator (C) is moved around toward the 20% position, it closes progressively, so directing a greater proportion of the air into the ether chamber.

The central chamber has a second inlet port leading from the ether vaporizing chamber via the thermocompensating valve (T). This port is fully closed in the "transit" position but opens progressively as the indicator is moved toward the 20% position, permitting extra air to leave the vaporizing chamber. The control mechanism thus directs progressively larger proportion of the air through the ether chamber without altering the total flow through the inhaler. It also seals the ether chamber when the inhaler is not in use. The temperature compensating mechanism is a sealed bellow-shaped capsule containing liquid ether which allows more air to pass through the vaporizing chamber when the temperature in the vaporizing chamber drops thereby, maintaining the volatile agent output concentration constant between 15° and 30°C. The ether capsule (bellows) should normally last for 10 years.

The indicator on the compensating unit, which shows red if the temperature is too high (over 32°C). At normal working temperatures (20–25°C), the metal top and black band will show. Mark III EMOs from 1964 do not have this feature. If the ether in the chamber becomes too hot (after storage in a hot environment), some ether must be vaporized by opening the filler and sucking air through with the bellows which has the effect of cooling the inhaler and will prevent a dangerously high concentration being delivered in the first few breaths. As ether vaporizes, heat is consumed at the rate of 89 cal/g of liquid.

When supplied, the water jacket is dry and must be filled with approximately 1200 mL of cold distilled water via the filler (F). Mark I EMOs have aluminum water jackets. Water level must be checked every 3 months because chemical action may lead to generation of gas which may damage other parts of the inhaler. Mark II, Mark III, Mark IV EMOs have stainless steel jackets and need only yearly water level checks. In freezing conditions, automobile anti-freeze solution containing 25% glycol should be used.

When filling the control must be turned to the zero position rather than to the "transit" position so that air can escape from the vaporizing chamber. As ether is poured in, the filler must be held down. Mark I inhaler have fillers, which must be lifted and turned. If left open by mistake, extra air enters the chamber and vaporizes too much ether. The inhaler must not be overfilled otherwise the wicks will be covered and vaporization of the ether will be decreased. 150 mL is required to fill it to the "Empty" mark (this being

the amount soaked up by the wicks), further 300 mL to raise the level to "Full."

An EMO requires no maintenance for 5 years. The main fault is that the control pointer may stick—this is most likely to occur if an inhaler is left for long periods full of ether without being used. It may also follow the use of ether dispensed from lacquered cans. Use of trichloroethylene or halothane, apart from corroding the metal of the interior, will also lead to the deposition of gummy material on the rotor, causing it to stick. If the unit is going to be stored for any length of time, drain the ether chamber and open the control fully to allow all the vapor to escape, otherwise oxidation of the ether will occur (Fig. 3).

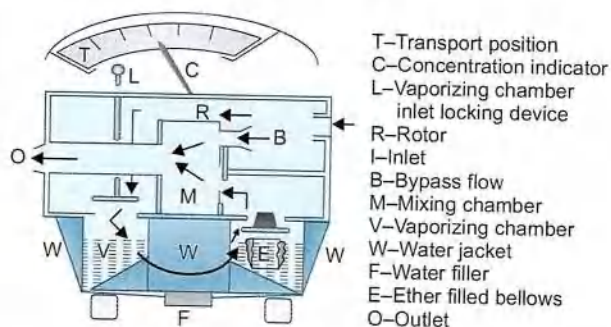


Fig. 3: Epstein-Macintosh-Oxford inhaler.

Q. What are the techniques of temperature compensation in EMO?

Ans. Temperature compensation in EMO occurs by:

- Supplied heat by the water jacket
- Flow alteration at the outlet of the vaporizing chamber via the ether bellows.

Q. How is the EMO checked?

Ans. The level indicator is checked by inverting the inhaler (with concentration control at "Transit"), when it should fall freely to the "Full" position. Connect the bellows to the inlet and block the outlet. While the control knob is turned to the "Transit" position, the filler held open, the bellows are compressed and no air should escape. The filler is released, the control pointer turned to 10% and this is repeated to check that the filler is not leaking. Bellows is then attached to the outlet, inlet is blocked, and the control knob is set at "2" to suck air through the safety release valve. If the valve is working correctly, air can be heard hissing through.

Q. Describe the Oxford miniature vaporizer (OMV).

Ans. This is designed for agents less volatile than ether, such as halothane, MOF, and trichloroethylene. In particular, it is meant as an induction unit for use with the EMO, when it should be plugged into the outlet of the latter. It should never be fitted to the inlet of the EMO, otherwise halothane will be drawn in, causing corrosion. It can be used with

a continuous gas flow for supplementation of relaxant anesthesia. When combined with an inflating device it can be used as a drawover anesthetic apparatus on its own to provide anesthesia or analgesia.

It is 13.5 cm high and weighs 1,060 g (with the water jacket full). There is a scale, which gives volumetric percentage concentration at 25°C with small deviations from the indicated concentrations with time and temperature. The output is never greater than indicated and is unaffected by positive pressure, so it is safe to place it on the patient's side of the bellows. Alternative scales are for halothane (HALO-4%), methoxyflurane (MOF-0.6%), and trichloroethylene (TCE-1.5%). The base contains a water jacket which acts as a heat source. When used with the EMO, the flow of gas through the vaporizer should be from the right to left (when looking at the front). Another version designed for gas machines in which the gas flows from left-to-right. The direction of flow is marked by an arrow.

The OMV has a special filler designed to limit the volume of anesthetic used. It is a useful economy device operated by a lever which must be pressed fully down to open the filling port. The initial movement of the lever opens an air relief valve; heavier pressure must be applied to open the actual filler. The built-in funnel surrounding the filler contains about 10 mL which is convenient for most induction purposes. It is enough to cover about one/eighth of the level indicator. A second 10 mL dose may be added initially if desired, but no more. The indicator is designed such that some liquid remains in the chamber even when it can no longer be seen in the window. The wicks are made of stainless steel gauze, which will not be corroded by any anesthetic in current use.

Q. What are the accessories for use with the EMO and OMV?

Ans.

Oxford Inflating Bellows

Oxford Inflating Bellows (OIB) is 3.5 kg and is 25 cm long by 23 cm high and 18 cm wide spring-loaded concertina bellows with two nonreturn flap valves. It was originally intended for use with a simple spring-loaded expiratory valve. This arrangement works well with spontaneous respiration; for artificial respiration, it is less satisfactory. For IPPV, a nonbreathing or inflating valve is usually preferred. Compression of the bellows during artificial respiration will produce full deflection of the valve flaps or bobbin during inspiration. In order to draw the bobbin or flaps back and hence to permit the patient to expire to atmosphere, a very small amount of air must pass back up the corrugated tube toward the bellows. If the distal bellows valve is not immobilized, air will be unable to pass back and the nonbreathing valve will stick in the

position of inspiration. A magnet is therefore provided for immobilizing the distal flap. The magnet must be fitted whenever a nonbreathing valve is used. The bellows have internal springs; press-studs inside the concertinas keep them closed during storage and transport. The bellows are capable of delivering a volume of about 1300 mL; a 10 cm stroke will deliver about 800 mL.

Penlon Bellows Unit (PBU)

This has a single-flap valve designed specifically for use with a nonbreathing valve. It must never be used with a simple spring-loaded expiratory valve.

A special pediatric bellows of smaller diameter has a full stroke capacity of about 400 mL. The EMO is also supplied as an alternative with the Ambu double-ended self-inflating bag. The bag is placed between the vaporizers and the nonbreathing valve.

Q. How is the EMO used with the anesthesia machine?

Ans. The flowmeter is plugged directly into the air inlet of the EMO. This arrangement can allow the EMO to operate as a plenum (constant gas flow) vaporizer, allowing a lower than indicated concentration of ether to be delivered if the fresh gas flow is <10 L/min. If the fresh gas flow is less than the patient's minute volume, air will still be drawn through the inhaler. Danger is that the bellows overfilling and a nonbreathing valve sticking if the fresh gas flow is greater than the patient's minute volume.

Q. What are the means of enriching the inspired mixture in an EMO with oxygen?

Ans. A tap for addition of oxygen is provided on the bellows, which is intended for use only in resuscitation and should always be kept closed during anesthesia. Oxygen is added to the inhaled air through a narrow delivery tube passed into the air inlet of the EMO. However, the best way of introducing oxygen is with the T-piece, gas reservoir adapter and the reservoir tube, which avoids the dilution of anesthetic vapor, which occurs when oxygen is led directly into the OIB or PBU concentration of oxygen achieved will depend both on the volume added and the patient's minute volume.

Q. How is the EMO used for artificial respiration?

Ans. By virtue of its bellows, the EMO apparatus can be used for artificial respiration in any situation. This applies equally to the OIB and the PBU. In either case, an automatic nonbreathing valve is essential. Alternatively, a self-inflating bag may be used. A magnet should always be used with the nonreturn valve (NRV) on the expiratory side of the OIB, to prevent sticking of the valve. When using a nonbreathing valve, it should be remembered that the movements of the bellows or bag effectively operate the valve. With most types of automatic nonbreathing valve,

it is possible by gentle pressure to blow air across the valve and out through the expiratory port instead of into the patient's lungs. In the case of Ruben valve, which has a hard plastic bobbin, the bobbin can be made to vibrate rapidly to and fro, making a shuddering noise. In either of these cases, ventilation would not be effective. The inspiratory movement should therefore begin smartly and continue throughout inspiration as a firm and definite compression of the bag or bellows. Similarly, at the beginning of expiration, it is possible to lift the bellows so slowly that the valve remains in the inspiratory position and air is allowed to pass back up the tubing toward the vaporizer, instead of out to the atmosphere. For this reason, inspiration should end, not with the bellows held down, but with a sudden upward movement, which is continued until the bellows has been refilled with air for the next breath. Similarly, the bag should be allowed to reexpand immediately. A pause for expiration must then be allowed.

The magnet on the OIB should never be used to immobilize the distal flap valve when a spring-loaded expiratory valve is used. Spring-loaded valves should never be used with the PBU. In either case, rebreathing into the bellows would occur.

Q. What are the principles for the arrangement of the EMO apparatus?

Ans.

- If vaporizers are to be placed in series, the vaporizer for the more volatile agent should be placed upstream (i.e., further from the patient) from the vaporizer for the less volatile. If this point is neglected, condensation of the less volatile agent (which will have a higher boiling point) will occur in a cooler downstream vaporizer. EMO should therefore always be upstream of the OMV. When using serial OMVs for halothane and trichloroethylene, the halothane OMV should be upstream.
- The bellows or inflating bag should be placed between the vaporizer and the patient. If air is blown through the vaporizer, the concentration delivered may be greater than indicated. This particularly affects the EMO because of its large internal volume, but OMV will not be affected, but it is nevertheless preferable to keep the vaporizer upstream. The exception is that when the PBU is used with the EMO and OMV, the bellows should be placed between the two vaporizers to avoid overbalancing them.
- Oxygen and nitrous oxide (when used) should always be introduced on the upstream side of the vaporizers. This avoids the dilution of the vapor, which will occur if the gas is introduced downstream. The oxygen inlets on the OIB and PBU are only intended for use in resuscitation.

- When using the OIB the magnet must be used to immobilize the downstream flap valve whenever an automatic nonrebreathing valve is used. This valve must not be immobilized if a simple spring-loaded expiratory valve is used.
- Before using the apparatus to anesthetize the patient, the bag or bellows must be operated in order to ensure that the flow of air is in the correct direction (i.e., toward the patient).

Q. How is the EMO used in pediatric patients?

Ans. In pediatric patients, the EMO cannot be used as a drawover vaporizer.

The dead space of the nonrebreathing valves and connections used for older children and adults is too high when used for infants and children below the age of 3 or 4 years. Moreover, the inspiratory flow rates generated by infants and very young children are too small for satisfactory vaporization of ether in the EMO. For these reasons, the techniques described for adults are not suitable and the EMO must be used as a constant-flow over vaporizer. The pediatric entrainer was designed to provide a flow of oxygen-enriched air through the EMO in order to vaporize the ether. It consists of fine jet, through which oxygen passes into a Venturi-shaped tube. Air is drawn in and mixes with the oxygen. The entrainer is plugged into the air inlet of the EMO.

Alternative means of providing a flow may be used. Nitrous oxide and oxygen may be used (5.0 L/min of oxygen and 5.0 L/min of nitrous oxide) which are delivered from a gas anesthetic machine via the EMO flowmeter attachment. Whichever source of gas flow is used, the EMO is used as a constant-flow or plenum vaporizer. In order to deliver the correct concentration, a total gas flow of about 10 L/min is needed.

The pediatric entrainer is driven from an oxygen cylinder, preferably via a regulator (reducing valve). The entrainer will deliver the correct flow of 10 L/min, if the flow of gas from the cylinder is increased until the manometer reads 100 mm Hg. By using oxygen as the driving gas, an inspired oxygen content of about 35% is achieved. Although this is clinically advantageous, overcoming most of the effects of ventilation-perfusion inequalities, it renders an ether mixture explosive. When compressed air is used as the driving gas, the ether remains flammable but is no longer detonable. For the infants, it is nevertheless safer to employ an oxygen-rich mixture to minimize the risk of hypoxemia developing as a result of the patient coughing or holding his breath. If the use of diathermy or open flames is essential, a non-explosive agent must be used instead of ether. The consumption of driving gas is 2 L/min. The entrainment ratio is 1.5:1. If the outflow from the entrainer is

occluded the high pressure gas escapes through the air inlet holes and the maximum internal pressure achieved is 15 cm H₂O, which is unlikely to harm the patient.

The entrainer is inserted into the air inlet of the EMO the bellows and OMV or Bryce-Smith Induction Unit (BSIU) is left in the circuit but the bellows is pushed down into the "closed" position. The anesthetic mixture is led to the patient via the delivery tube to T-piece, one end of which leads to the patient via a facemask or endotracheal tube. The other end has a reservoir tube, which should have a volume greater than the patient's tidal volume. Too short a tube will result in air-dilution. The delivery tube of the Ayre's circuit is attached to the outlet of the OIB in place of the long corrugated tubing. When the Penlon bellows is used, the OMV or BSIU remains on the downstream side of the bellows. The delivery tube is then attached to the outlet of the accessory vaporizer.

The minute volumes of anesthetized children are found to be 1.5 L in a 1-year-old child, 2 L in a 3-year-old, and 3 L in a 5-year old. A fresh gas flow of 2.5–3 times the minute volume should be used with Ayre's T-piece to prevent rebreathing of the expired gas. The flow delivered by the entrainer is therefore adequate for the largest child likely to be anesthetized by this method. The possible disadvantage of using a flow greater than three times the minute volume is that the resistance to expiration will be increased. A flow of 10 L/min may be unnecessarily high for neonates but the advantage of knowing the concentration of ether outweighs this disadvantage. The peak expiratory flow in a child under 1 year will be <6.6 L/min. The maximum flow in the expiratory limb of the T-piece will therefore be 16.6 L/min. Therefore, with a T-piece of 10 mm diameter as recommended by Ayre, the resistance is likely to be negligible.

Q. Classify and describe Goldman's vaporizer.

Ans.

Classification

- Variable by pass, drawover or flowover, can be used with or without wick, in- or out-of-system
- No temperature compensation
- Multiple agent.

Model

- **Mark I:**
 - Self-locking in off position
 - Three divisions between off and on position.
- **Mark II:**
 - Click stops at each settings
 - Three divisions between off and on position
 - Size and shape of ports differs from Mark I.

- **Mark III:**
 - Two divisions between off and on position
 - Click at each setting.

Construction

It consists of:

- Small glass bowl which will hold up 20 cc liquid.
- Bowl is attached to head which receives gas and divides gas between vaporizing chamber and by pass.
- A control lever on the top is used to alter the vaporizer output and is turned counterclockwise to increase the concentration.

Evaluation and Use

- Mainly it is designed for attachment to the outlet of an intermittent flow machine.
- Low-resistance vaporizer.
- Easy to operate and does not deliver high-concentration.
- When vaporizing chamber is full, output concentration is 2%.
- It is used inside a breathing circle, it maintains fairly constant halothane concentration.

Care and Cleaning

- Drained periodically.
- Vaporizer chamber should be cleaned out and allowed to dry.

Hazards: Agitation or tilting will increase the vapor output.

Table 2 shows the halothane percentages in Goldman's vaporizer.

TABLE 2: Halothane percentages in Goldman's vaporizer.

Drum position	Gas flow rate		
	2 L/min	8 L/min	30 L/min
1	0.03	0.03	0.03
2	0.41	0.74	0.92
3	0.73	2.21	1.31
On	0.74	2.08	1.21

Q. How can the output of Goldman's vaporizer be increased?

Ans.

- Agitation or splashing from filling markedly increases halothane concentration to over 5%. However, this can lead to liquid agent entering the breathing circuit, and is hence not recommended.
- By adding wicks—concentration is increased between 3% and 4% at III setting.
- Two Goldman vaporizers in series, turning one to the third position and altering the setting of other to increase vapor output.

Q. Classify and describe copper kettle.

Ans.

Classification

- Measured flow
- Bubble through
- Out of system
- Temperature compensated by supplied heat and manual flow alteration
- Multiple agent.

Construction: Each copper kettle is supplied with a vaporizer circuit control valve and a flowmeter assembly. Copper is used in construction because of its high heat capacity and thermal conductivity.

Gas flow: Gas enters the vaporizer from the inlet and passes upward through the central tube into the surge chamber or loving cup. This chamber prevents the sudden surge of gas into the vaporizer. The gas then passes downward around the center tube and enters the diffuser at the base of the vaporizer. There is a sintered bronze disc at the top of the diffuser. The disc conducts heat to the gas-liquid interface to prevent cooling. As the carrier gas permeates through the disc, it is broken into small bubbles, which rise through the anesthetic liquid. This increases the area for saturation with vapor. Gas saturated with vapor rises to the top of the kettle and passes out through the discharge tube. A thermometer is present to indicate the vaporizer temperature. A level indicator window is present. The filling port was on top of the vaporizer in older models, and is at the back in newer ones. Newer models also have a back pressure check valve at the outlet. The copper kettle is available in two sizes with capacities of 160 and 400 mL. A chart is available to indicate the flow needed through the vaporizer as per agent, temperature and required output.

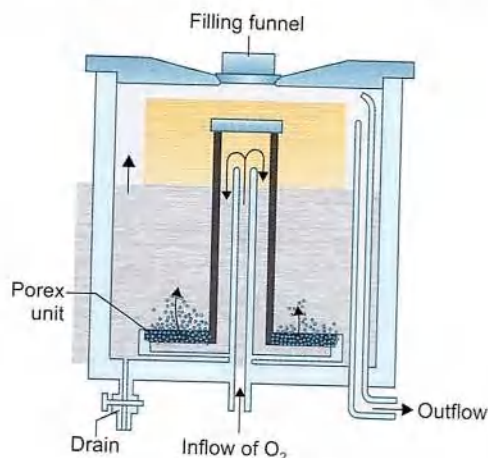


Fig. 4: Copper kettle.

Hazards: Calculations are needed to determine the flow of gas needed through the vaporizer for a particular vapor output. Erroneous calculations can lead to lethal doses being delivered. Failure to turn on the main gas flow can result in undiluted vapor delivery to the patient. When filled from the top, older models could be overfilled leading to spillage into the delivery tube. This is eliminated in newer models by changing the filling port to the side (**Fig. 4**).

Q. Classify and describe TEC vaporizer (TEC 1 to 5) temperature compensated vaporizers—TEC.

Ans.

Classification

1. Variable bypass
2. Flowover with wick
3. Out of system
4. Temperature compensated by automatic flow alteration
5. Concentration calibrated
6. Agent specific.

Functioning of TEC 2, 3, 4, 5, and 7 Vaporizers

The gases pass through the vaporizer by two channels, one leading through a bypass and the other through the vaporization chamber. The proportion of gases passing through the bypass is determined by a calibrated control knob that is attached to a spindle. In the off position, the vaporizing chamber is isolated from the bypass and all gases flow through the bypass to the fresh gas outlet. When the spindle is pulled toward the operator and rotated counterclockwise, the inlet and outlet ports of the vaporizing chamber are opened. The fraction of gas passing through the vaporizing chamber is also varied by a bimetallic thermostatic valve. This is located inside the vaporization chamber in TEC 2 vaporizers, but is in the bypass chamber in subsequent models. As the temperature in the vaporization chamber falls, the thermostatically-operated valve opens wider and a greater proportion of the gas flow passes through the chamber. By this means, the vapor output is kept constant. The vaporization chamber contains a series of wicks, with a large surface area. This ensures that gases exiting the vaporization chamber are saturated with vapor.

TEC 1

TEC 1 is introduced by Cyprane Company in 1956 as Fluotec I for halothane. All Mark I vaporizers were recalled, modified, and rereleased as Mark II variants in order to correct a critical design flaw, which could cause the proportioning valve to stick thus posing a risk of over dosage to the patient.

TEC 2

- Halothane—Fluotec—Mark II
- Methoxyflurane—Pentec—Mark I.

Construction: Parts

- Vapor chamber is round and wicks are concentric and in series
- Filling tap at side
- Draining at bottom
- Level indicator on side
- External calibrated control knob is attached to a spindle that controls concentration—moves to right
- Temperature compensation—bimetallic strip located at the outlet of vaporizing chamber and regulates the amount of gas that passes through the chamber.

Evaluation and use:

- Not accurate below 4 L/min
- With flows of 2 L/min, at dial setting <2%, output is less than setting; with dial setting >2%, output is more than the setting
- With nitrous oxide, it gives higher output at lower setting and lower output at higher setting
- With dial setting between off and 0%, some output can occur, varying with gas flows
- Prone to back pressure changes—pumping effect at low flows and pressurizing effect at high flows
- There can be a small leak in the off position.

Care and cleaning:

- Manufacturers recommend yearly return to company for servicing.
- *Halothane*: Preservative is thymol which accumulates in the vaporizer.
- Vaporizer is drained every 2 weeks.
- *Drained agent*: Discarded.

Hazards:

- Thymol caused the operating spindle to stick
- Back pressure forcing saturated vapor back into the bypass chamber
- High concentration at low flows.

TEC 3**Models:**

- Fluotec Mark III
- Pentec Mark II
- Enflurtec
- Fortec.

Construction:

It consists of two sections:

1. Lower vaporizing chamber
2. Upper duct and valve system.

Single control dial: It controls the concentration delivered by this vaporizer. Movement of this dial opens and closes appropriate parts and thus regulates the proportion of gas passing through vaporizing chamber.

Parts:

- This model has larger bypass.
- The tube leading to the vaporizing chamber is longer and has an expansion area.
- Wicks are excluded from inlet area of vaporizing chamber—helps to decrease effects of back pressure on vaporizer output.
- Concentration dial is on top and to the left of it is the locking lever which is depressed to turn on the vaporizer.
- Sight window for liquid level on left.
- Filling and draining at the bottom.

Evaluation and use:

- All are accurate with lower dial setting.
- At higher dial setting, higher than expected concentration at low flow rates and lower than expected concentration at high flow rates.
- Sudden increase or decrease in carrier gas flow, intermittent back pressure and upstream O₂ flush has negligible effect on vapor output.
- Nitrous oxide has got little effect on output.
- Between off and 0.5% dial setting, output was less affected by fresh gas flows, as compared to TEC 2.
- Small amounts of leaks in bypass in off position.
- Tipping up to 90° does not affect output. Tipping >90° causes an increase in subsequent output.

Care and cleaning:

- Manufacturer's annual preventative maintenance
- Halothane to be drained—every 2 weeks.

Hazards:

Besides common hazards, problems related to dial rotation have been reported:

- Rotated beyond off position
- Rotated by 180°
- Leak from dial setting because of damage or compression of gasket.

TEC 4**Models:**

- Fluotec (IV)
- Enflurtec (IV)
- Fortec (IV).

Construction: This is a modified version of TEC 3. These vaporizers are designed to be attached to the back bar of anesthesia machine by means of Select-a-Tec manifold which allows easy removal and exchange of vaporizer.

Parts:

- On top is a control dial that is turned counterclockwise to increase the concentration. A release button located to the left of the concentration dial must be depressed before the vaporizer can be turned on.
- There is a locking lever connected with the control dial, so that the vaporizer cannot be turned on unless it is locked to the manifold.
- When the vaporizer is turned on, two plungers within the vaporizer operate to open the valve into the fresh gas stream. Also, when the vaporizer is turned on, two extension rods prevent the use of any adjacent vaporizer.
- There are two filling mechanisms—a screw cap or a keyed filling system. A special adapter fitted the bottles of the specified agent only, while the other end was keyed for specific same agent vaporizers.

Gas flow:

- Off position—incoming gas flows from inlet through bypass and on to outlet.
- On position:
 - Incoming gas is split into two streams by a rotary valve attached to the concentration dial
 - One stream is directed to the vaporizing chamber. That gas first enters one of two chambers, which surrounds the bypass chamber. After passing from this chamber, it is directed over two concentric wicks, which enclose a copper helix, which converts this space into long spiral outlet channel. Wicks assure maximum contact between gas and agent. The vapor-laden gas leaves via the second chamber surrounding bypass chamber to outlet.
 - The remaining fresh gas flows to bypass chamber, which contains the temperature sensitive element.
 - No spillage after tilting or inversion.

Evaluation and use:

- Fluctuating back pressures can increase the concentration by altering the flow distribution within the vaporizer.
- Greatest effects are observed at:
 - Low flow rates
 - Low dial setting
 - Large and frequent pressure fluctuation.
- Output decreases slightly when nitrous oxide is used as a carrier gas.
- Vaporizer is filled and used in upright position—deviations do not affect the output or safety, but can give a misleading impression of the amount of agent in the vaporizer.

Care and cleaning:

- Annual servicing
- When liquid level is low, drain it and discard it
- Every 2 weeks, vaporizer is drained.

Problems with TEC 4:

- Difficulty in operation one-handed
- Yearly service interval.

*TEC 5**Models:*

- Isotec (5)
- Fluotec (5)
- Enflurtec (5)
- Sevotec (5).

Construction:

- At the top is the control dial
- Release button at rear of dial is pushed in before vaporizer is turned on
- Rear end is locking lever connected to the dial so that vaporizer cannot be turned on until it is locked
- Filling devices—keyed filling system and screw cap.

Differences:

- Helical intermittent positive pressure assembly to minimize effects of positive pressure ventilation.
- Volatile agent capacity increased from 125 to 300 mL.
- One-handed dial control and more obvious “off” position.

Evaluation:

- Accuracy is maximum at flow of 5 L/min with dial setting <3%
- Decreased output with higher flows with higher setting
- TEC 5 is more prone for increasing vapor output due to pumping effect than TEC 4
- Carrier gas composition affects the output. If air or nitrous oxide is used, the vaporizer output is lower than the setting at low flows, and higher than the setting at high flows
- Reverse flow through the vaporizer increases output.

Maintenance:

- Every 2 weeks or when there is low level of liquid, the vaporizer needs to be drained
- Every 3 years, servicing by company.

Q. Enumerate differences and improved safety features between TEC 2 and 5.

Ans. Differences and improved safety features between TEC 2 and 5 are described in **Table 3**.

TABLE 3: Differences and improved safety features between TEC 2 and 5.

	TEC 2	TEC 3	TEC 4	TEC 5
Flow (L/min)		0.25–15	0.25–15	0.25–15
Temperature (°C)		18–35	18–35	18–35
Capacity (mL)				
Dry wicks	150	270	135	300
Wet wicks	50	35	100	225
Concentration dial	At side	On top	On top	On top
Range (%)	Off–4%	0.5–5.0	0.5–5.0	0.5–5.0
Weight (kg)		6.3	7.2	7.0
Temperature compensation	Yes, at outlet of vaporizing chamber		Yes, in bypass chamber	
Nonspillage	No	No	Yes	Yes
Tilt	No	90°	180°	180°
Interlock	No	No	Yes	Yes

Q. Why does desflurane need a separate kind of vaporizer?

Ans. Desflurane has certain unique properties, which preclude its use in regular vaporizers:

- Its vapor pressure is higher than other volatile agents. At 20°C, the vapor pressure of desflurane is 669 mm Hg. Therefore, larger quantities of desflurane are present in the vapor form in the vaporizing chamber. Therefore, much larger quantities of fresh gas flow through the bypass chamber would be needed to dilute this vapor to clinically meaningful concentrations using a traditional mechanical bypass. For example, at 1 atm and 20°C, 100 mL/min of gas through the vaporizing chamber picks up 735 mL of desflurane as compared to 47 mL of halothane. To dilute this to a 1% concentration, 73 L/min of bypass flow would be needed for desflurane as compared to about 5 L/min for halothane. Modern Aladin vaporizer is electronically controlled and therefore a separate vaporizer is not needed for desflurane.
- Desflurane boils at 22.8°C. Therefore, at room temperatures >22.8°C, the amount of vapor formed would be limited only by the heat available from the vaporizer.
- The MAC of desflurane is higher than that of other modern inhaled anesthetics (6–7%). Therefore, the amount of desflurane needed over a period of time is larger than other anesthetics. This would cause cooling of the vaporizer, and a reduction in output, which cannot be compensated by the temperature compensating mechanisms available in contemporary vaporizers. Therefore, an external heating source is needed.

Q. Classify and describe TEC 6 vaporizer.

Ans. The TEC 6 vaporizer for desflurane is classified as:

- Concentration calibrated
- Thermocompensated (electrically supplied heat)
- Agent-specific
- Out of circuit
- Plenum
- Gas-vapor blender.

The vaporizer has two independent circuits arranged in parallel. Fresh gas flows through one circuit, through a fixed resistor and emerges at the fresh gas outlet. Desflurane vapor flows through the other circuit through a pressure regulating valve. There is a pressure transducer between these two circuits which transmits the pressure difference to a control electronic system. This regulates the pressure regulating valve so that the pressure in the two circuits, is equaled (known as the working pressure) even if the fresh gas flow is changed. A distal variable resistor in the vapor circuit allows concentration of gas to be adjusted by the operator. The vapor mixes with the fresh gas at the outlet of the vaporizer (Fig. 5).

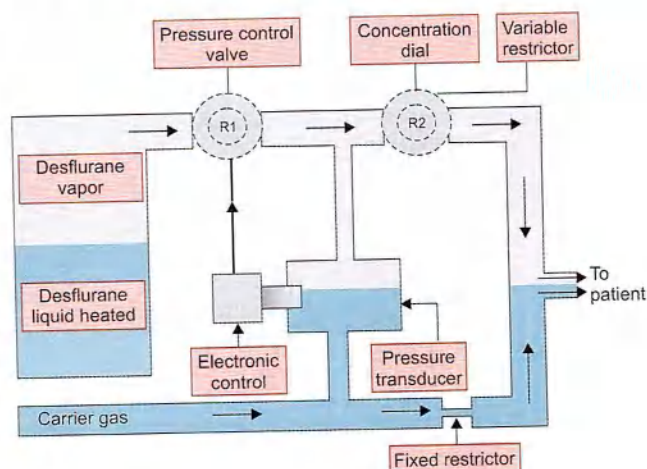


Fig. 5: Working of desflurane vaporizer.

The TEC 6 vaporizer has a concentration dial at the top, which is calibrated from 1–18% (1% graduation till 10% and 2% till 18%). The dial release is at back and is depressed to start the vaporizer, and when concentrations higher than 12% are needed. There are separate filling and draining mechanisms. There is a front display panel for visual indicators for vaporizer function. This consists of a light-emitting diode. The various color-coded alarms are:

- Amber—without audible alarm—warm up, along with audible alarm—low agent level below 50 mL
- Green—operational status
- Red—no output—due to:
 - Low agent level < 20 mL
 - Power failure

- Malfunction
- Tilted vaporizer beyond 10°
- A liquid-crystal display (LCD) indicates the amount of liquid in the vaporizer:
 - Once the vaporizer is connected to an electrical source, the vaporizer sump gets heated to 39°C. During this warm-up phase, the concentration dial is in standby mode and the vaporizer cannot be used. Once the proper temperature is achieved, the green light comes on indicating readiness for operation.

The TEC 6 vaporizer cannot be classified as a measured flow or a variable-bypass vaporizer since it does not follow traditional methods of vaporization. It is best classified as a gas-vapor blender. Some authors have described it as an injector, though this term is ideally used for the technique of injection of liquid agent into the fresh gas flow.

Maintenance: Servicing every year.

Q. Describe some of the newer vaporizers.

Ans.

Aladin 2™ Cassette (GE Health Care)

Aladin 2™ Cassette is available for isoflurane, sevoflurane, and desflurane for use with the Aisys Carestation™. Cassettes are color coded and have a magnetic code for automatic agent detection. These agent-specific cassettes operate with innovative electronic control from the host anesthesia delivery system. The anesthetic agent control consists of two parts: the electronic control mechanism in the anesthesia system and the separate Aladin 2 agent Cassette.

Agent concentration is adjusted by regulating the amount of fresh gas flowing through the cassette. A proportional valve is used to regulate the flow. Part of the fresh gas bypasses the cassette, so the more fresh gas allowed to pass through the cassette, the greater the concentration.

Fresh gas flow along with flows in and out of the cassette, is electronically measured for enhanced control. Each Aladin 2 Cassette is magnetically coded, allowing the Aisys Carestation to recognize which type of agent cassette is inserted.

Advantages

- Can be stored in any position
- Light-weight; can be carried around easily
- Automatic record-keeping and gas usage calculation.

TEC 7

- Similar to TEC 5
- "Easy-Fil" filler mechanism
- New ergonomics and design
- Planned factory service free
- Simplified filler mechanism

- Soldered sump assembly eliminating seals
- Improved sight glass design
- Clear agent color identification.

Q. Describe injection type vaporizers. How are they different from conventional variable bypass vaporizers?

Ans. Injector type vaporizers are measured flow type of vaporizers. Maquet 950® and Drager DIVA® are currently available in Maquet FLOW i and Drager Zeus anesthesia workstations respectively.

The external appearance is different as they have a filling port but do not have a concentration dial. The vaporizers are agent specific.

Variable bypass vaporizers split the fresh gas flow, part of which goes through the vaporizing chamber and the rest goes to the bypass chamber. The proportion of fresh gas flowing through vaporizing chamber, i.e., the carrier gas is controlled mechanically, e.g., in TEC 5, 7, etc. or electronically, e.g., in Aladin vaporizer. The splitting ratio is determined by the dial setting, fresh gas flow rate and temperature compensation. The vaporization chamber is also the reservoir of volatile anesthetic agent where vaporization occurs to a saturated vapor pressure.

Achieving the saturated vapor pressure and having an accurate splitting ratio is paramount to the efficiency of the conventional variable bypass vaporizer. TEC 7, 5, and earlier versions are mechanically controlled with no active feedback mechanism. TEC 6 and Aladin vaporizers have pressure sensors in the vaporizers which along with other electronic devices such as flow sensors control the vaporizer output making them more accurate.

In an injection type vaporizer, the amount of volatile agent needed to achieve a particular agent concentration is calculated based on electronic information of the desired agent concentration and fresh gas flow and the vapor. The liquid agent is first metered and a calculated dose is then vaporized. The vapor is then injected into the patient circuit. Sensors and relief valves ensure accuracy and safety. Thus there is no carrier gas which picks up the vapor. Agent concentration monitoring is done downstream of the vaporizers for accuracy.

Factors which make the injection vaporizers more accurate:

- Metering of dose in liquid phase makes it more accurate
- Carrier gas flow rate and composition does not affect performance
- Temperature and ambient pressure also do not affect performance
- Moreover, agent monitoring increases accuracy and safety of the vaporizer.

Both the Maquet and Drager vaporizers have similar features:

- Reservoir of volatile anesthetic agent from where a microprocessor controlled small amounts of liquid agent is pushed into the vaporizing chamber.
- A heated chamber vaporizes the liquid which is then added to the fresh gas flow or the patient circuit.
- Agent concentration is measured in the outflow tract for accuracy.
- No risk of tipping or spilling.

In Drager Zeus anesthesia workstation, in a closed loop feedback system, the desired end-tidal agent concentration is set. Based on pharmacokinetic and pharmacodynamics models, the required inspired agent concentration is computed. Based on this and the fresh gas flow, the liquid agent is injected into the vaporizing chamber followed by its addition into the breathing circuit in a controlled manner. This helps in achieving rapid changes in agent concentration in a closed loop system. Also there is efficient use of volatile anesthetic agent.

Q. Describe the use of vaporizers in hyperbaric and hypobaric conditions.

Ans. Vaporizers are calibrated at normal atmospheric pressures (760 mm Hg). At an atmospheric pressure of 500 mm Hg, SVP of halothane would be 243 mm Hg. Therefore, halothane vapor in the vaporizing chamber would contribute to $243/500 = 48\%$ volume and the remaining 52% volume will be constituted by carrier gas. From the previous example, we have seen that the splitting ratio for 1% halothane at a fresh gas flow rate of 5 L is 106:4,844. Therefore, 106 mL of carrier gas would enter the vaporizing chamber. This will account for 52% volume and will pick up $106/52 \times 48 = 98$ mL of halothane vapor.

Therefore, the gas exiting the vaporizing chamber would be 106 mL of carrier gas + 98 mL of halothane vapor. This would combine with 4,844 mL of bypass flow. The final concentration of halothane would be 98 mL in a total volume of $98 + 106 + 4,844 = 98/5,048 = 1.9\%$.

This is almost twice the 1% concentration achieved at atmospheric pressure.

The partial pressure of halothane will be $1.9/100 \times 500 = 9.5$ mm Hg. This is only slightly higher than its partial pressure at atmospheric pressure.

Therefore, at hypobaric conditions, variable bypass vaporizers deliver a higher concentration in volumes percent, but the partial pressure increases only slightly. Since the anesthetic properties of a gas are determined by its partial pressure, there is no significant clinical implication. Similarly, in hyperbaric conditions, delivered concentration decreases significantly but partial pressure reduces only slightly.

The new concentration (c') achieved at altered ambient pressure (p') can be given by the formula:

$$c' = c \times p/p'$$

where c is the concentration set on the dial and p is the sea-level pressure

The TEC 6 vaporizer works on the principle of differential pressure transduction. Therefore, when ambient pressures decrease or increase, the volume percent delivered will remain constant; however, this volume percent now represents a lower or higher partial pressure respectively, because of the change in ambient pressure. Hence, there will be a change in clinical effect.

Q. What is the effect of change in carrier gas on vaporizer output?

Ans. Vaporizers are calibrated with 100% oxygen as the carrier gas. When the carrier gas is switched from 100% oxygen to 100% nitrous oxide, there is a transient fall in vaporizer output, followed by an increase to a new steady-state level. This transient decrease is due to increased solubility of nitrous oxide in the volatile agent, leading to a reduction in the amount of gas leaving the vaporizing chamber.

Q. What should be the arrangement of vaporizers on the manifold?

Ans. Old anesthesia machines had up to three variable-bypass vaporizers arranged in series such that fresh gas from the flowmeters passed through each vaporizer to reach the common gas outlet of the anesthesia machine.

Without the use of an interlock, it was possible to turn on all vaporizers simultaneously. This could lead to:

- Overdose of anesthetic
- Vapor from one vaporizer could condense in the downstream vaporizer leading to contamination.

To prevent this, various techniques of arranging vaporizers have been described:

- Vaporizers of agents with lower boiling points should be upstream and those with higher boiling points should be downstream to prevent condensation.
- Vaporizers are present with less potent agents upstream and more potent agents downstream, so that even if there is contamination, it does not have clinical implications.
- Difficult-to-clean vaporizers should be placed upstream.
- Agents with toxic byproducts (like trilene) should be placed downstream.

Modern vaporizers are equipped with interlocking devices, which allow flow of gas through only one vaporizer at a time, and prevent simultaneous operation of more than one vaporizer.

SUGGESTED READING

1. Davis PD, Parbrook GD, Kenny GNC. Basic Physics and Measurement in Anaesthesia, 4th edition. Oxford: Butterworth-Heinemann; 1995.
2. Dorsch JA, Dorsch SE. Understanding Anaesthesia Equipment, 2nd edition. Baltimore: Lippincott Williams and Wilkins; 1984.
3. Dorsch JA, Dorsch SE. Understanding Anaesthesia Equipment, 4th edition. Baltimore: Williams and Wilkins; 1998.
4. Farman JV. Anaesthetic vapour concentrations in the EMO system. *Anaesthesia*. 1984;39:171-80.
5. Miller RD, Eriksson LI, Fleisher LA, Wiener-Kronish JP, Cohen NH, Young WL. *Miller's Anaesthesia*, 8th edition. Philadelphia: Churchill Livingstone; 2015.
6. *Ward's Anaesthetic Equipment*, 2nd edition. London: Bailliere Tindall Publishers; 1993.

Anesthesia Breathing Systems

Sheila Nainan Myatra, Parmanand Jain

Q. How do you define breathing system?

Ans. A breathing system is an assembly of components which connects the patient's airway to the anesthetic machine creating an artificial atmosphere, into and from which the patient breathes.

Q. What are the requirements of a breathing system?

Ans.

Essential:

- Deliver the gases from the machine to the alveoli in the same concentration as set
- Delivery of gases in the shortest possible time
- Effective elimination of carbon dioxide
- Minimal apparatus dead space
- Low resistance.

Desirable:

- Efficient for spontaneous as well as controlled ventilation
- Adaptability for use in adults and children
- Economy of fresh gas
- Conservation of heat
- Adequate humidification of inspired gas
- Light weight and convenient to use
- Provision to reduce theater pollution.

Q. What are the common components of a breathing system?

Ans.

- A delivery tube through which the fresh gases are delivered from the machine to the system and a fresh gas entry port
- Breathing tubes
- Reservoir bag
- Pressure relief valve
- Sleeves, connectors, adapters
- CO₂ absorber (only if rebreathing is to be allowed)
- Vigilance aids (e.g., FiO₂ sensor), humidification system may be present.

Q. What is the function of the reservoir bag? How does it function as a reservoir?

Ans.

- Serves as a reservoir of gases during exhalation, which is available for next inspiration
- Reservoir of gases to provide peak inspiratory flow during spontaneous breathing
- Permits rebreathing and allows greater economy of anesthetic gases and prevents air dilution
- To give a manual assisted or controlled breath
- Visual and tactile assessment rate and depth of respiration during spontaneous breathing
- Tactile assessment of resistance and compliance during manual assisted breathing
- Buffer for an increase in airway pressure in the system
- To provide a manual "sigh".

Q. What are adjustable pressure-limiting (APL) valves? Describe the Heidbrink valve.

Ans.

- These are expiratory valves, which provide control of pressure in the breathing system.
- The purpose of this valve is to allow the escape of exhaled air and surplus gases from the breathing circuit, but without permitting entry of air from outside during a negative pressure in the system.
- Usually, it is desirable that the pressure required to open the valve must be as low as possible in order to minimize resistance to expiration. It must however present sufficient resistance to prevent the reservoir bag from emptying spontaneously.

Heidbrink valve: This is a commonly used expiratory valve. The components are shown in **Figure 1:**

- Female taper
- Retaining screw
- Disk
- Spring
- Valve top.

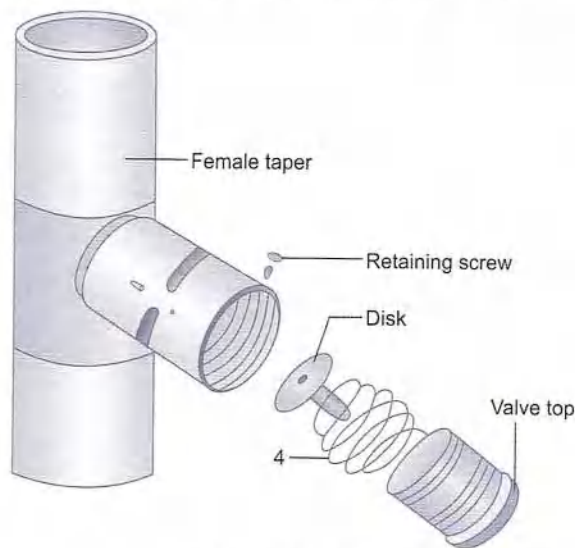


Fig. 1: Heidbrink valve.

The body of the Heidbrink valve has a female taper with a retaining screw. The valve disk is very light and rests on a knife edge seating, which presents a small area of contact. This reduces the adhesion between the disk and the seating due to the surface tension of the condensed water from expired air. The disk has a stem which is located in a guide in order to ensure correct positioning on the seating and a coiled spring of light weight which promotes closure of the valve. The spring is a delicate coil and is of such dimensions that when the valve top is fully screwed "open" there is minimal pressure on the valve when seated. However, during the "blow off" phase the disk rises and shortens the spring so that the pressure exerts on the disk is greater and will close it at the appropriate time. Screwing down the valve top produces progressively increasing tension in the spring. When the top is fully screwed down, the valve is completely closed. This metal valve is constructed so that it is easy to dismantle; to be cleaned and sterilized.

Q. What are the ideal characteristics of tubing used in a breathing circuit? Describe and compare the corrugated rubber tube with the plastic disposable tubing.

Ans. Ideal characteristics of tubing used in a breathing circuit:

- The tubing in a breathing system must be of such diameter as to present a low resistance to gas flow.
- Its cross section must be smooth and uniform in order to promote laminar flow where possible.
- It should be flexible and should not kink.
- It should drape easily so that a loop may hang between the patient and the apparatus, which can trap moisture and prevent it from going back to the apparatus.
- Transparency is desirable.

The corrugated rubber tubing is 22 mm in diameter and 110 cm in length. When compared with the plastic corrugated tubing, the advantage of the corrugated rubber tubing is that its ends are more easily stretched so it makes a better union with the other components of different diameters. It is also reusable. The disadvantage is that it is not transparent and it is heavier than the plastic tubing. Its irregular walls cause much turbulence and it may harbor dirt and infection. Recently available silicon tubes have combined advantage of plastic tubes (transparent with smooth inner walls) and corrugated rubber tubes (nonkinkable with easily stretchable ends) and are available in various sizes of 110, 130, and 150 cm.

Q. What is the significance of setting the fresh gas flow (FGF) and how is it set for the effective elimination of CO₂ in the breathing system with bi-directional flow?

Ans.

Significance of setting the fresh gas flow:

- This is a very essential component of the breathing system with bidirectional flow. If there is no FGF the patient will suffocate.
- It is also required for the effective elimination of CO₂ and to adjust the amount of rebreathing.
- If the FGF is excessive, there will be wastage of gases.
- If a system has to deliver a set concentration of gases to the patient in the shortest time the FGF should be as near the patient as possible.

Elimination of carbon dioxide: An example to understand CO₂ elimination by the bidirectional flow systems is as follows. Normal production of carbon dioxide in a 70 kg adult is 200 mL/min and it is eliminated through the lungs. Normal end-tidal concentration of carbon dioxide is 5%. Hence to eliminate 200 mL of carbon dioxide as a 5% gas mixture, the alveolar ventilation has to be:

$$\frac{200 \times 100}{2} = 4,000 \text{ mL} = 4\text{L}$$

This 4 L is the normal alveolar ventilation. Any breathing system should provide a minimum of 4 L/min of carbon dioxide free gas to the alveoli for eliminating carbon dioxide. If the alveolar ventilation becomes <4 L/min, it would lead to hypercarbia. If the alveoli are ventilated with 5 L/min of a gas containing 1% carbon dioxide, or 8 L/min of a gas containing 2.5% carbon dioxide, it could still eliminate 200 mL of carbon dioxide per minute from the alveoli.

Q. What is rebreathing? What are the factor affecting rebreathing and what are the effects of rebreathing?

Ans. Rebreathing is said to occur when the expired alveolar gas containing CO₂ (and less oxygen than normal) is inspired as part of the next tidal volume. Circuits should have minimal rebreathing.

Rebreathing depends on:

- Fresh gas flow rate (when the inspired volume is more than the FGF)
- Design of the breathing system
- Mode of ventilation (spontaneous or controlled)
- Patients' respiratory pattern (tachypnea will increase rebreathing)
- Mechanical dead space.

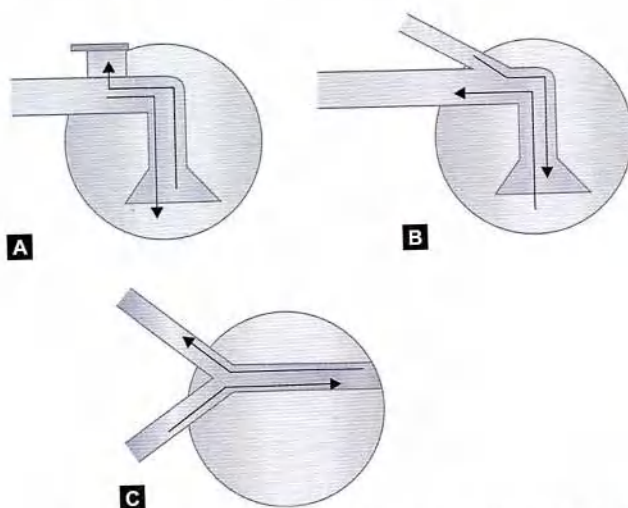
Effects of rebreathing:

- Retention of heat and water
- Alteration of inspired gas concentration
- Elevation of blood CO₂ levels.

Q. What is patient and apparatus dead space?

Ans. Patient dead space: Patient dead space is that part of the respiratory system that is occupied by air that does not take part in gas exchange. In an adult, the volume of this space is 150 mL, but varies with the movement of the lungs during respiration. In a normal patient, this will mean only the anatomical dead space under ideal conditions:

- *Anatomical dead space* consists of the air passages from the lips or the nares up to, but not including the alveoli.
- *Physiological dead space* includes in addition to the anatomical dead space air that does not take part in the respiratory exchange, i.e., air in the nonperfused alveoli (dead space ventilation).



Figs. 2A to C: Apparatus dead space. (A) In an afferent system—up to the expiratory valve; (B) In an efferent system—up to the FGF; and (C) In a system with separate inspiratory and expiratory limbs (e.g., circle absorber) up to the point of bifurcation.

Apparatus dead space: This is the volume in the anesthetic equipment that is filled with exhaled gases at the end of expiration. In the nonrebreathing system, this volume will be reinhaled in the next breath and in the closed system this volume would be reinhaled without passing through the CO₂ absorber. In other words, it is the volume of the

breathing system from the patient-end to the point up to which, to and fro movement of expired gas takes place. Simplest definition of apparatus dead space is from the patient-end to the point where inspired and expired gases meet and are separated. For example, apparatus dead space extends from the patient-end of the circuit up to the following points (when the FGFs are adequate) in the examples given in **Figures 2A to C**.

Rebreathing of apparatus dead space does not contribute to either heating or humidification, but can reduce alveolar ventilation.

Q. What are the causes of a discrepancy between the inspired and delivered concentration of gases?

Ans.

- Rebreathing
- Leaks in the system leading to air dilution
- Dilution of gases because of repeated activation of flush valve
- Uptake of anesthetic agent by the system
- Release of anesthetic agent by the system.

Q. What are the different ways of classifying circuits? How do you classify circuits by function?

Ans.

Circuits can be classified in many ways: Dripps et al. have classified them as *open, semi-open, semi-closed, and closed*, taking into account the presence or absence of reservoir, rebreathing, CO₂ absorption and directional valves (**Tables 1 and 2**).

TABLE 1: Classification of circuits by function.

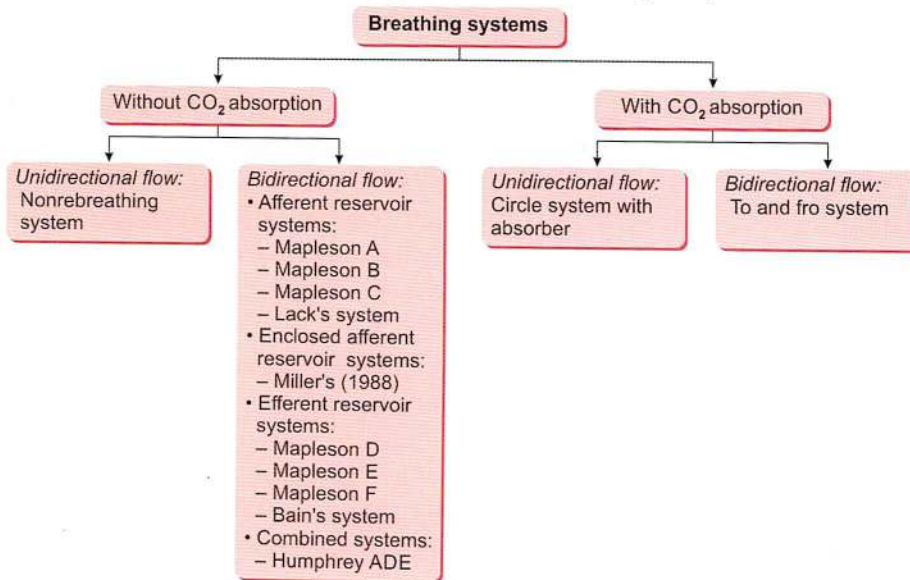
Mode	Reservoir bag	Rebreathing	Example
Open	No	No	Open drop
Semi-open	Yes	No	Nonrebreathing circuit, or circle at high FGF (> minute ventilation)
Semi-closed	Yes	Yes, partial	Circle at low FGF (< minute ventilation)
Closed	Yes	Yes, complete	Circle [with pop-off (APL) valve closed]

(APL: adjustable pressure-limiting; FGF: fresh gas flow).

TABLE 2: Rebreathing and nonrebreathing circuits.

Parameters	Rebreathing	Nonrebreathing
Valve	Spill (APL)	Unidirectional
Access of expired gases to reservoir bag	Yes	No
Closed reservoir bag	+	±
Volume emerging from valve	Equivalent to FGF	Equivalent to MV

(FGF: fresh gas flow; MV: minute ventilation).

Flowchart 1: Breathing system with and without CO₂ absorption.

Breathing systems with and without CO₂ absorption: The ambiguity of the terminology used as *open*, *semi-open*, *semi-closed* and *closed* allowed inclusion of apparatus that are not breathing systems at all into the classification (Flowchart 1).

To overcome this problem Conway suggested that a *functional classification* be used and classified according to the method used for CO₂ elimination. Miller DM in 1988 widened the scope of this classification to include the enclosed afferent reservoir (EAR) system.

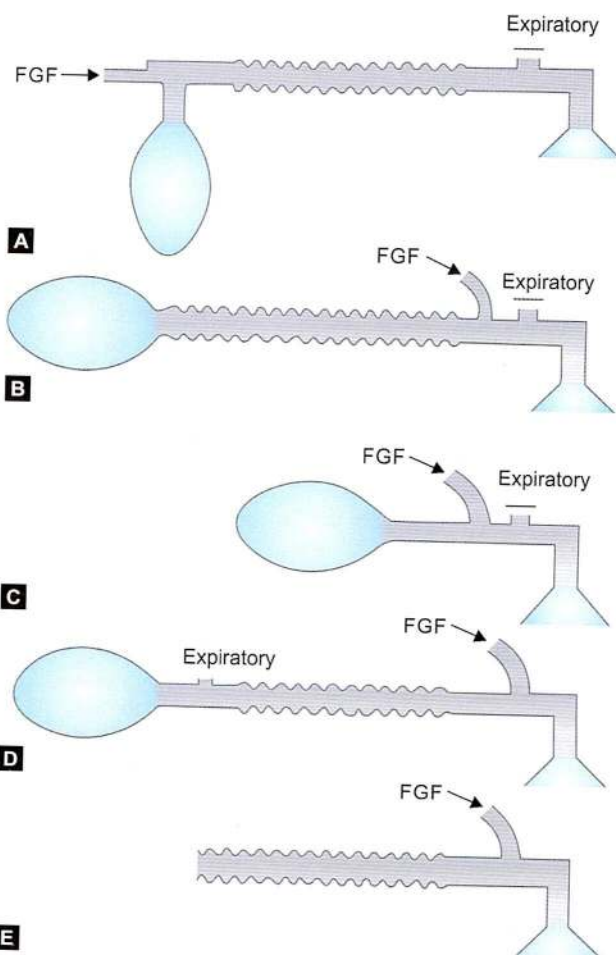
Q. Draw diagrams of Mapleson's classification of semi-closed breathing systems. Which are the afferent and efferent reservoir (ER) systems? Rank the Mapleson breathing systems according to their superiority both for controlled and spontaneous ventilation.

Ans. Mapleson in 1954 classified the semi-closed anesthetic system according to the position of the expiratory valve, the FGF inlet and the position/presence of the reservoir bag. These are Mapleson's systems: A, B, C, D, E (no provision for CO₂ absorption). The Jackson-Rees modification of the Ayre's T-piece is classified as a Mapleson F system although it was not included in the original description by Professor Mapleson (Flowchart 1 and Figs. 3A to E).

Afferent reservoir systems: They have the reservoir in the afferent limb and do not have an efferent limb—Mapleson A, Mapleson B, and Mapleson C.

The afferent systems work efficiently during spontaneous breathing provided the expiratory valve and the reservoir bag and FGF are separated by one patient tidal volume; and the apparatus dead space is minimal. They are not very efficient for controlled ventilation. In Mapleson B and C, the FGF is close to the expiratory valve, hence the system

is inefficient both during spontaneous and controlled ventilation.

**Figs. 3A to E:** Mapleson breathing systems (FGF: fresh gas flow).

Efferent reservoir systems: They have valves or breathing bags at the expiratory end of the circuit—Mapleson D, Mapleson E, and Mapleson F.

They act as T-pieces with the FGF delivered to the patient end of the circuit. These systems are all inefficient for spontaneous respiration.

At present only systems A, D, E, and F and some of their modifications are used.

Ranking of Mapleson's systems based on superiority is as follows:

1. *Spontaneous ventilation:* A > D, F, E > C, B
2. *Controlled ventilation:* D, F, E > B, C > A.

Q. What are the advantages and disadvantages of the Mapleson's breathing systems?

Ans.

Advantages:

- Simple
- Light weight
- Not bulky, light weight and convenient to use
- Inexpensive parts
- Low resistance
- Low dead space
- All parts can be easily cleaned and sterilized.

Disadvantages:

- High FGF, hence wasteful and costly
- Elimination of CO₂ depends on FGF so there can be rebreathing if FGF inadequate
- Loss of heat and humidity (except in coaxial circuits).

Q. What are the assumptions made by Mapleson when he analyzed bidirectional flow systems?

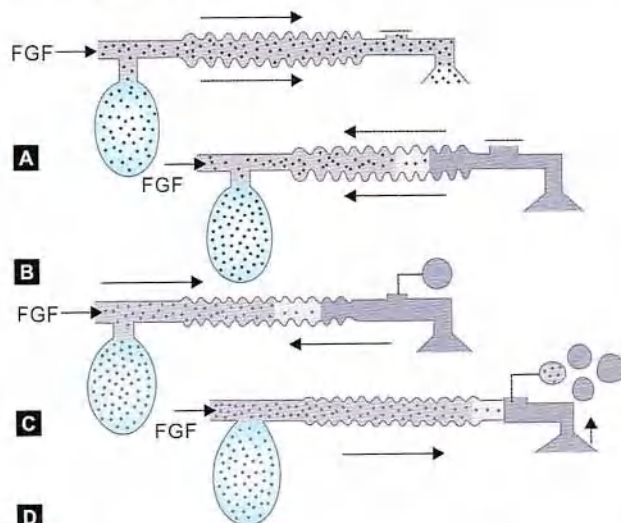
Ans.

- Gases move en bloc. They maintain their identity as fresh gas, dead space gas, and alveolar gas. There is no mixing of these gases.
- The reservoir bag continues to fill up, without offering any resistance till it is full.
- The expiratory valve opens as soon as the reservoir bag is full and the pressure inside the system goes above atmospheric pressure.
- The valve remains open throughout the expiratory phase without offering any resistance to gas flow and closes at the start of the next inspiration.

Q. Describe Mapleson A breathing system and give the functional analysis during spontaneous breathing and controlled ventilation. What are the advantages and disadvantages of this system?

Ans. The Mapleson A (Magill) system was designed by Sir Ivan Magill in the 1930s and remains an excellent system for spontaneous ventilation. Fresh gas enters the system at the fresh gas outlet of the anesthesia machine. It consists of a reservoir bag, a corrugated hose (42", volume that of

one tidal volume, about 550 mL), and an expiratory valve (Heidbrink valve) which is very close to the patient to reduce the dead space.



Figs. 4A to D: Functional analysis during spontaneous breathing in Mapleson A circuit (FGF: fresh gas flow).

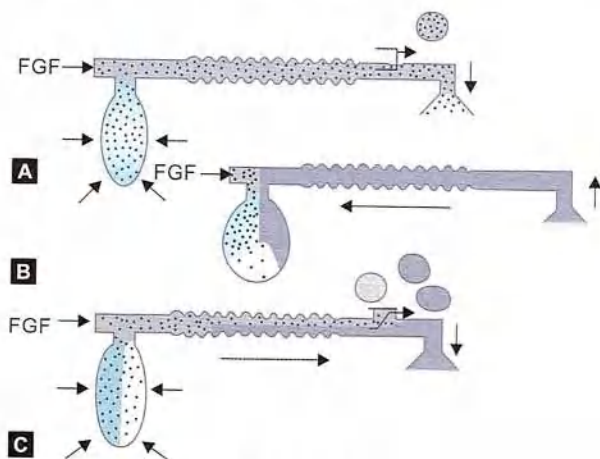
Spontaneous breathing (Figs. 4A to D): During inspiration, gas is inhaled from the 2 L reservoir (breathing) bag which partially collapses giving a visual confirmation that breathing is occurring (A). During expiration the bag and tubing are initially refilled with a combination of exhaled dead space gas (containing no carbon dioxide) and fresh gas flowing from the anesthesia machine (B). Once the bag is full the pressure within the breathing system rises and the expiratory valve near the patient opens allowing the alveolar gas (containing carbon dioxide) to be vented from the system (C). During the expiratory pause, more fresh gas enters the system driving any remaining alveolar gas back along the corrugated tubing and out through the valve (D).

If the FGF is sufficiently high all the alveolar gas is vented from the circuit before the next inspiration and no rebreathing will take place. With careful adjustment the FGF can be reduced until there is only fresh gas and dead space gas in the breathing system at the start of inspiration. When the system is functioning correctly, without any leaks, a FGF equal to the patient's minute ventilation is sufficient to prevent rebreathing. Thus, a FGF closer to the patient's total minute ventilation (including dead space) is usually selected to provide a margin of safety. An adult's minute volume is approximately 80 mL/kg/min and thus for a 75 kg man a FGF of 6 L/min will prevent rebreathing. This is an efficient system for spontaneously breathing patients if carbon dioxide absorption is not available.

The opening pressure of the Heidbrink valve must be low to prevent expiratory resistance (0.5 cmH₂O), however, it must be higher than the collapsing pressure of the reservoir

bag (0.25 cmH₂O) otherwise the system will not work during spontaneous breathing.

Controlled ventilation (Figs. 5A to C): During controlled ventilation, the Magill circuit works in a different way and becomes wasteful and inefficient, requiring high FGF to prevent rebreathing. The inspiratory force is provided by the anesthetist squeezing the reservoir bag after partly closing the expiratory valve next to the patient. During lung inflation, some of the gas is vented from the circuit and at the end of inspiration the reservoir bag is less than half full (A). During expiration, dead space and alveolar gas pass down the corrugated tubing and may reach the bag which will then contain some carbon dioxide (B). During the next inspiration when the bag is compressed, alveolar gas reenters the patient's lungs followed by a mixture of fresh, dead space, and alveolar gas (C).



Figs. 5A to C: Functional analysis during controlled ventilation in Mapleson A circuit.

A FGF of two and a half times the patient's minute volume is required to vent enough alveolar gas to minimize rebreathing (FGF of about 12–15 L/min) which is obviously very inefficient. In practice, the Magill circuit should not be used for positive pressure ventilation except for short periods of a few minutes at a time. Prolonged use is dangerous.

Advantages:

- Best among all Mapleson's systems for spontaneous breathing
- Minimal wastage of gases during spontaneous breathing. No rebreathing if FGF is about one minute volume.

Disadvantages:

- Not efficient for controlled ventilation—considerable rebreathing requires high FGF.
- Expiratory valve required—produces slight resistance during expiration.
- Expiratory valve is heavy (Heidbrink valve).

- Expiratory valve near patient end inconvenient to use especially when head and neck is draped and difficult for scavenging.
- Not suitable for pediatric use.

Q. Enumerate the modifications of the Magill's system and describe the Lack circuit.

Ans.

Lack's Circuit

This is a simple modification of the Mapleson A circuit which made it more efficient for controlled ventilation. This is achieved by substituting a nonrebreathing valve (such as the Laerdal's valve) for the Heidbrink valve at the patient end of the circuit. Not only does this arrangement prevent rebreathing, but during manual ventilation the delivered minute volume will also be the same as the desired FGF. It is, however, a dangerous arrangement for spontaneous respiration because the valve may jam if the FGF is greater than the patient's minute volume.

Lack's coaxial system: Another modification of the Magill's system originally described in 1976. A disadvantage of the Magill system is that the expiratory valve is attached close to the patient making it awkward to use. The Lack's system is a coaxial version of Mapleson A system with the expiratory valve located near the machine end toward which the exhaled gases are carried by efferent tubing placed coaxially. This also facilitates easy scavenging of expired gas. This system functions like a Mapleson A system both during spontaneous and controlled ventilation. The FGFs required for both spontaneous and controlled ventilation are as described for the standard Mapleson A system. The dead space of the apparatus is from the patient end up to the expiratory valve. The *advantages* of this modification are the easy accessibility of the expiratory valve (it is near the machine end unlike the Magill's circuit), easy to scavenge and better separation of gases before the APL valve is reached (**Fig. 6**).

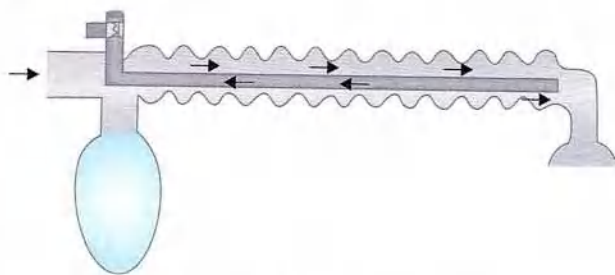


Fig. 6: Lack's coaxial system.

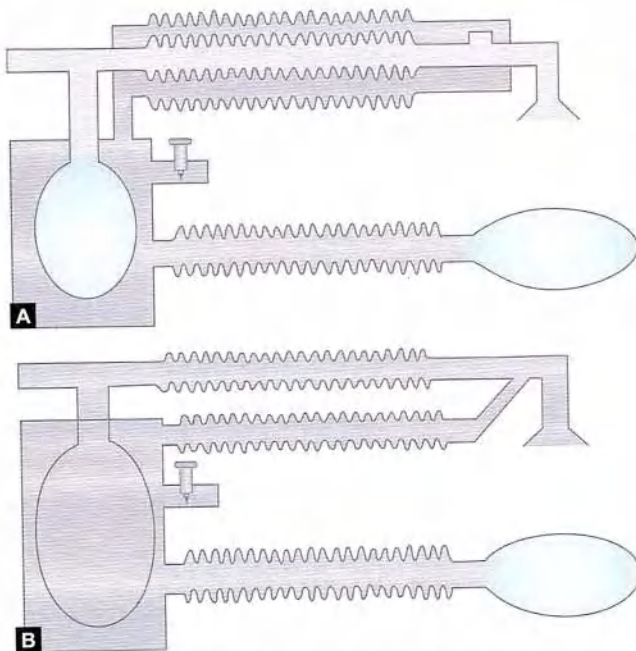
Testing the Lack's Coaxial Circuit:

- Attach the endotracheal tube to the patient end of the circuit and blow through it after closing the valve fully. If there is a leak in the circuit, the bag will move.

- Close both the limbs at the patient end. Open the expiratory valve and squeeze the bag. If there is a leak the bag will collapse and the gas will escape through the valve.

Enclosed afferent reservoir system: This system consists of a Mapleson A system; enclosed within a nondistensible structure, and it was described by Miller and Miller in 1998. It may also be constructed by enclosing the reservoir bag alone in a bottle and connecting the expiratory port to the bottle with a corrugated tube and a one way valve (B). To the bottle is also attached a reservoir bag and a variable orifice for providing positive pressure ventilation (**Figs. 7A and B**).

This system allows for the selective elimination of alveolar gases in both spontaneous and controlled ventilation. A comparison with the Bain system in controlled ventilation demonstrated greater efficiency in eliminating carbon dioxide. A FGF of 70 mL/kg/min using an EAR system gave mild hypocarbia which equated to a FGF of 100 mL/kg/min using the Bain system. Smaller minute volumes of ventilation are required for optimal performance than with the Bain system. It is thus more efficient than the Bain system for controlled ventilation.



Figs. 7A and B: Enclosed afferent reservoir.

Q. Describe Mapleson B and C system.

Ans. These systems are all functionally similar and also similar in construction, with the FGF entry and the expiratory valves located at the patient end of the circuit. The C system differs from B in that it does not have the corrugated tube. In order to reduce the rebreathing of alveolar gas and to improve the utilization of FG during

controlled ventilation, the FG entry was shifted near the patient. However, since this allows a complete mixing of FG and expired gas these systems are not good for spontaneous or controlled ventilation. Fresh gas flows should be at least 2–3 times the minute volume to minimize rebreathing. Hence, they are not commonly used in anesthetic practice, although the C system is used on intensive care units. High flows of gases are needed to prevent rebreathing of CO_2 . This system was at one time combined with a canister of soda lime to absorb CO_2 (Waters' "To and Fro" Circuit).

Q. Describe and draw Ayre's T-piece? What is the Mapleson E system?

Ans. This was described by Phillip Ayre in 1937. This consists of a light metal tube 1 cm in diameter, 5 cm in length with a side arm. It functions as a nonrebreathing system (**Fig. 8**).

The Mapleson E is a modification of the Ayre's T-piece. Fresh gas enters the system through the side arm and the expired gas is vented into the atmosphere through the expiratory limb, which also serves as a reservoir (there is no bag in this system). An expiratory limb volume greater than the patient's tidal volume prevents entrainment of room air and thereby prevents dilution of anesthetic gases. A FGF greater than three times the minute ventilation prevents rebreathing. The dead space is minimal as it is only up to the point of FGF entry. Controlled ventilation is given by intermittently occluding the outlet with the thumb. The advantage of this system is that it has a low dead space, no valves and a low resistance, which makes it very suitable for use in children. The disadvantage of this system is that the pressure buffering effect of the bag is absent and we cannot make a visual and tactile observation of respiration. This system requires 2–3 times the minute ventilation to prevent rebreathing.

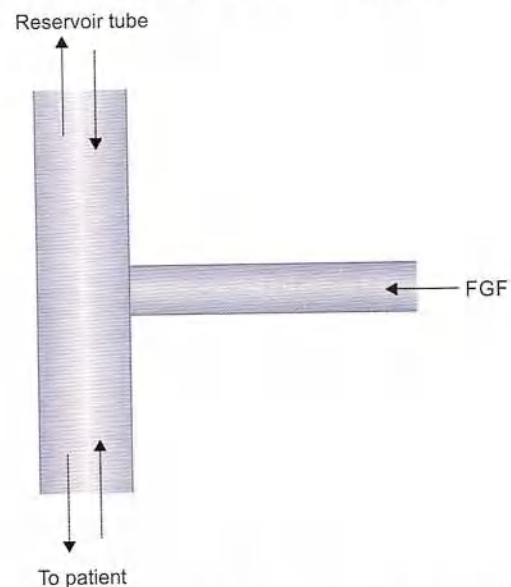


Fig. 8: Ayre's T-piece (FGF: fresh gas flow).

All the efferent systems are modifications of the Ayre's T-piece.

Q. Describe the efferent reservoir systems?

Ans. The Mapleson D, E, and F systems are ER systems; they are all modifications of the Ayre's T-piece. They have a 6-mm diameter tube as the afferent limb that supplies the FGF from the machine. The efferent limb is a wide-bore corrugated tube to which the reservoir bag is attached and the expiratory valve is near the bag. In Mapleson E system, the corrugated tube acts as the reservoir. In Bain system, the afferent and efferent limbs are coaxially placed.

In an attempt to reduce FGF requirements, ER systems are constructed with reservoirs in the efferent limb. The functioning of all these systems is similar. These systems work efficiently and economically for controlled ventilation as long as the FGF entry and the expiratory valve are separated by a volume equivalent to at least one tidal volume of the patient. They are not economical during spontaneous breathing.

Q. What is the Mapleson D system?

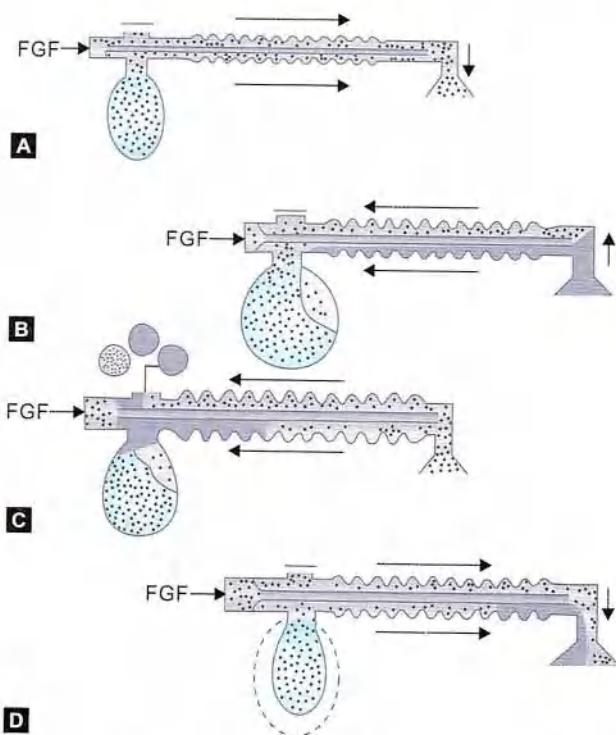
Ans. The Mapleson D can be described as a T-piece with an expiratory limb. The fresh gas inlet is located near the patient end and the expiratory valve is present, which is close to the reservoir bag. The functional analysis is described with the Bain's circuit which is a modification of Mapleson D system.

Q. Draw and describe the Bain's circuit. Give the functional analysis of this system during spontaneous breathing and controlled ventilation.

Ans. The Bain's circuit is the most commonly used modification of the Mapleson D system. It is a coaxial circuit, which was introduced in 1972 by Bain and Spoerel. This is often called a universal circuit as it can be used for adult and pediatric patients as well as for spontaneous and controlled ventilation. It is also the best among the Mapleson circuits for controlled ventilation. This is made of plastic tubings, which are arranged coaxially which reduces the bulk of the system. The inner tubing is green in color with an internal diameter of 7 mm through which the FGFs to the patient and exhaled gases travel in the outer corrugated tubing with an internal diameter of 22 mm. The reservoir bag may be removed and replaced by a ventilator. It also has a pressure manometer and PEEP valve. The dead space of the circuit is the volume from the patient end up to the point of separation of the gases. If there is a leak in either tubing, the entire volume of the tubing becomes the dead space.

Spontaneous respiration: Before connecting to the patient breathing system should be filled with FG. When the patient inspires, the FG from the machine, the reservoir bag and the corrugated tube flow to the patient (A). During expiration, there is a continuous FGF into the system at the patient

end. The expired gas gets mixed with the FG as it flows back into the corrugated tube and the reservoir bag (B). Once the system is full, the excess gas is vented through the valve situated at the end of the corrugated tube near the reservoir bag. During the expiratory pause, the FG continues to flow and fill the proximal portion of the corrugated tube while the mixed gas is vented through the valve (C). During the next inspiration, the patient breaths FG as well as the mixed gas from the corrugated tube (D). The composition of the inspired mixture is determined by several factors. They are FGF, respiratory rate, tidal volume, end expiratory pause, and CO_2 production in the body. Factors other than FGF cannot be manipulated in a spontaneously breathing patient. It has been shown that the FGF should be at least 1.5–2 times the patient's minute ventilation in order to minimize rebreathing (Figs. 9A to D).



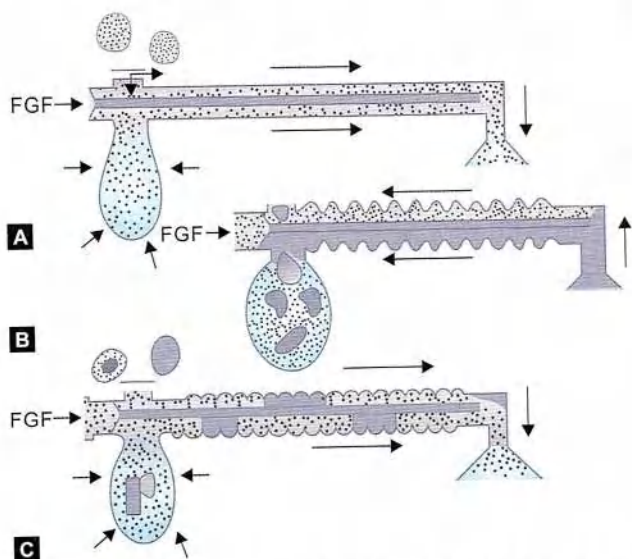
Figs. 9A to D: Functional analysis of Bain's circuit during spontaneous breathing (FGF: fresh gas flow).

Controlled ventilation: For positive pressure ventilation, the expiratory valve has to be partly closed so that it will open only after sufficient pressure has developed in the system. When the system is filled with fresh gas, the patient gets ventilated with the FGF from the machine, the corrugated tube, and the reservoir bag (A). During expiration, the expired gas gets mixed with the fresh gas that is flowing into the system at the patient end. During the expiratory pause, the FG will continue to enter the system and pushes the mixed gas toward the reservoir (B). When the next inspiration is initiated, the

patient gets ventilated with the gas in the corrugated tube, i.e., a mixture of FG, alveolar gas, and dead space gas (C). When the pressure in the system increases, the expiratory valve opens and the contents of the reservoir bag are released. This system functions more efficiently when used for controlled ventilation. The degree of rebreathing that occurs depends on the FGF. This system causes less rebreathing than Mapleson B and C (Figs. 10A to C).

Flow settings:

- *Controlled ventilation:* 2 L/min < 10 kg, 3.5 L/min = 10–50 kg, 70 mL/kg > 60 kg.
- *Spontaneous ventilation:* 200–300 mL/kg.



Figs. 10A to C: Functional analysis of Bain's circuit during controlled ventilation (FGF: fresh gas flow).

Q. What are the advantages and disadvantages of Bain's circuit? What are the tests that can be performed to check the Bain's circuit?

Ans.

Advantages:

- *Universal circuit:* It can be used for adult and pediatric patient. Spontaneous and controlled ventilation.
- Best Mapleson system for controlled ventilation.
- Light weight and coaxial arrangement makes it convenient to use.
- Long length of the circuit makes it easy to be away from the patient, useful during MRI.
- Disposable circuit, however can be easily sterilized and reused.
- Warmth added to the inhaled gases by exhaled gas passing through the outer tubing.

Disadvantages: Due to the coaxial nature of the circuit, there may be disconnection or kinking of inner tubing or a leak may get missed. If this occurs, the entire corrugated tubing

becomes dead space. This can result in hypercarbia from inadequate gas flow or increased respiratory resistance.

Tests: A careful examination of the Bain's circuit is essential. Special tests to check this circuit are given below:

- *The Pethick test:* Flush high flow oxygen into the circuit and occlude the patient's end of the circuit until the reservoir bag is filled. The patient end is then opened and the circuit flushed with oxygen. If the inner tube is intact, the Venturi effect occurs at the patient end, causing decrease in pressure within the circuit and the bag will deflate. If there is a leak in the inner tube FG will escape into the expiratory limb and the bag will inflate.
- Block the inner tube at the patient end and flush the circuit. The flow meter bobbins will dip due to the back pressure if the inner tube is patent and there is no leak in the inner tube.

Q. What are coaxial circuits? Describe the Penlon coaxial circuit?

Ans. Coaxial circuits have two tubings one inner and one outer tubing (arranged coaxially). This makes the system less bulky and convenient to use and also helps to warm the inspired gases (Fig. 11).

Examples of coaxial circuits are:

- Lack's circuit (described earlier)
- Bain's circuit (described earlier)
- Penlon circuit.

Penlon Circuit

It is a modification of the Bain's circuit. Here the inner tube is made of antistatic rubber and the outer tube is transparent. The circuit is directly connected with the outlet of the anesthetic machine by means of a coaxial circuit valve. Fresh gas inflow occurs through this valve unit into a narrow metal tube the end of which connects the inner tube of the circuit. This valve unit thus directs the FGF, forms an expiratory valve for exhaled gases and an attachment for the reservoir bag. The resistance in this system (at a flow rate of 30 L/min) is <0.5 cmH₂O.

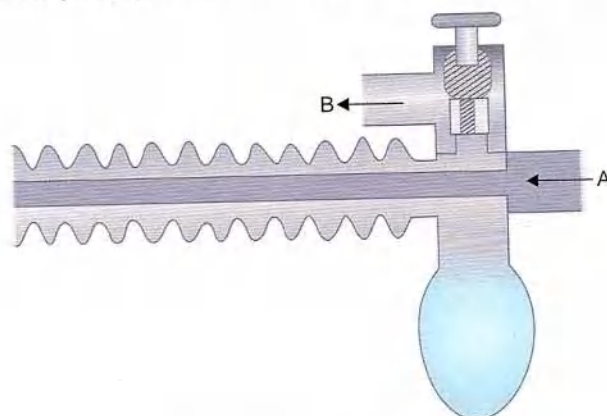


Fig. 11: Penlon circuit.

Q. Draw and describe the Jackson-Rees circuit in detail. What are its advantages and disadvantages?

Ans. Jackson-Rees modification of the Ayre's T-piece: This is classified as a Mapleson F system although it was not included in the original description by Professor Mapleson. An open-ended reservoir bag is fitted to the expiratory limb of the Ayre's T-piece. This reservoir bag incorporates a relief mechanism for venting exhaled gases. The relief mechanism is either an adjustable valve at the distal end of the bag or a fenestration at the side of the bag. The internal volume of the tube between the patient and the bag should exceed the patient's tidal volume. Movement of the bag can be seen during spontaneous breathing, and the bag can be compressed to provide manual ventilation (**Fig. 12**). It is light in weight, has low resistance and no valve which makes it suitable for use in children. As in the Bain's circuit, the bag may be replaced by a mechanical ventilator designed for use with children. This system is suitable for children under 20 kg. During spontaneous ventilation, exhaled gases pass down the expiratory limb and mix with the fresh gas. The expiratory pause allows the fresh gas to push the exhaled gas down the expiratory limb, including the reservoir bag. Fresh gas flows of 2–3 times minute volume should be used to prevent rebreathing during spontaneous ventilation, with a minimum flow of 3 L/min, e.g., a 4-year-old child weighing 20 kg has a normal minute volume of 3 L/min and would require a FGF of 6–9 L/min.

During controlled ventilation in children, normocapnia can be maintained with a FGF of 1 L + 100 mL/kg/min, e.g., a 4-year-old weighing 20 kg would need a total FGF of around 3 L/min. It can be used in adult patients with controlled ventilation using FGF ranging from 70–100 mL/min/kg body weight.

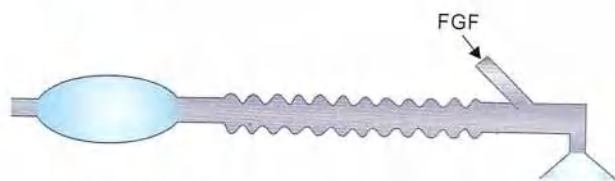


Fig. 12: Jackson-Rees circuit (FGF: fresh gas flow).

Advantages:

- Simple, easy to assemble
- Light weight
- Portable
- No valves
- Least resistance
- Suitable for pediatric anesthesia, especially head and neck surgery (due to the above factors)
- Easy to scavenge
- Inexpensive

- Equally effective for both controlled and spontaneous ventilation.

Disadvantages:

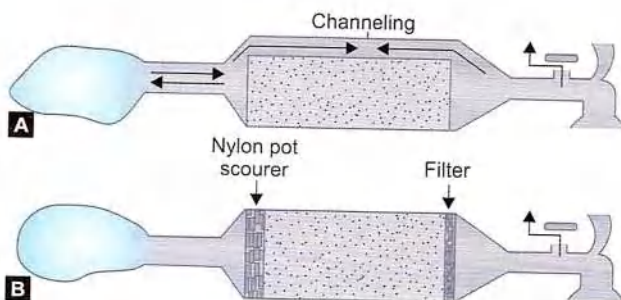
- Wastage of gases—FGF three times minute volume required
- Lack of humidification (can be overcome by allowing FG to pass through a humidifier)
- Occlusion of the relief valve can increase airway pressure producing barotrauma.

Q. Classify and describe Water's to and fro canister. What are the advantages and disadvantages of this?

Ans. This is a breathing system with CO₂ absorption with bidirectional flow. Here the patient breathes in and out of a closed bag, which is connected to the facemask or an endotracheal tube. The dead space is the part between the patient and the soda lime and hence this volume must be minimized, which means the soda lime canister must be close to the patient's head and this leads to mechanical problems. A length of tubing can be placed between the canister and the bag without detriment. The fresh gases enter the system at the patient end and the expiratory valve is between this point and the canister (**Figs. 13 and 14**).



Fig. 13: Water's to and fro canister.



Figs. 14A and B: Schematic diagram of Water's to and fro canister.

The canister is usually placed horizontally for convenience and it is important that it is well-packed with soda lime, since if there is a void above the soda lime "channeling" (gas takes the path of least resistance) would occur. The soda lime may be conveniently compressed to prevent gaps by the insertion of a nylon pot scourer at one

end. When the canister is closed the sealing washer should be checked to ensure that it is in the correct position. Any soda lime on the threads or the washer should be removed. The system should be checked for leaks. Cotton wool filters may be used at the patient end of the canister to prevent inhalation of soda lime.

Advantages:

- Portable
- Easy to sterilize after an infected case (if made of metal-autoclave, if made of Perspex-chemical sterilization).

Disadvantages:

- Cumbersome
- Risk of patient inhaling soda lime
- Danger of accidental extubation due to the weight of the canister and proximity to the patient
- Expiratory valve very close to patient end, difficult to use when head and neck are draped.

Q. What are combined breathing systems? Describe in brief Humphrey's ADE system.

Ans. Combined breathing systems are those in which more than one Mapleson's systems are combined and used. This helps to overcome the difficulties of changing the breathing systems for different modes of ventilation.

Humphrey's ADE System

The Mapleson A circuit is inefficient for controlled ventilation as is the Mapleson D circuit for spontaneous ventilation. David Humphrey has designed a system called Humphrey ADE, with two reservoirs, one in the afferent limb and the other in the efferent limb. This consists of a single circuit that can be changed from a Mapleson A system to a Mapleson D by moving a lever on the metallic block, which connects the circuit to the fresh gas outlet on the anesthetic machine, while in use, only one reservoir will be in operation. The reservoir bag is situated at the fresh gas inlet end of the circuit, and gas is conducted to and from the patient down the inspiratory and expiratory limbs of the circuit. It can be used for adults as well as children. The functional analysis is the same as Mapleson A in afferent system mode and as Bain in efferent system mode. It is not yet widely used.

Depending on the position of the control lever at the Humphrey block, gases either pass through the expiratory valve or the ventilator port. When the lever is "up" the

reservoir bag and the expiratory valve are used, creating a Mapleson A type circuit. When the lever is in the "down" position the bag and valve are bypassed and the ventilator port is opened creating a Mapleson D system for controlled ventilation. If no ventilator is attached and the port is left open the system will function like an Ayre's T-piece (Mapleson E).

It is essential that the anesthetist fully understands the function of a particular circuit. If the lever on the Humphrey block is moved from "up" to "down" while gases are flowing the breathing bag will remain full of gas but manual ventilation of the patient's lungs by compressing the bag will be impossible and may resemble complete obstruction of the breathing circuit. This has led to anesthetists occasionally concluding that their endotracheal tube required changing.

SUGGESTED READING

1. [online] Available from <http://www.capnography.com/Circuits/breathingcircuits1.html> [Last accessed February, 2020].
2. Conway CM. Anaesthetic breathing systems. *Br J Anaesth.* 1985;57:649-57.
3. Dorsch JA, Dorsch SE. The breathing systems II. In: Dorsch JA, Dorsch SE (Eds). *Understanding Anaesthesia Equipment, Construction Care, and Complications*, 3rd edition. Baltimore: Williams and Wilkins; 1994.
4. Dorsch JA, Dorsch SE. The breathing systems II. The Mapleson system. In: Dorsch JA, Dorsch SE (Eds). *Understanding Anaesthesia Equipment, Construction Care and Complications*, 2nd edition. Baltimore: Williams and Wilkins; 1984.
5. Miller DM, Miller JC. Enclosed afferent reservoir breathing systems. Description and clinical evaluation. *Br J Anaesth.* 1988;60:469-75.
6. Miller DM. Breathing systems for use in anaesthesia. Evaluation using a physical lung model and classification. *Br J Anaesth.* 1988;60:555-64.
7. Miller DM. *Textbook of Anaesthesia*, Vol.1, 4th edition.
8. Milner Q. *Anaesthetic Breathing Systems*. Update *Anaesth.* 1997;7:4.
9. Nuffield Department of Clinical Neurosciences. (2020). Divisions: Nuffield Division of Anaesthetics. [online] Available from http://www.nda.ox.ac.uk/wfsa/html/u07/u07_013.htm [Last accessed February, 2020].
10. The elimination of rebreathing in various semi-closed anaesthetic systems. MAPLESON WW. *Br J Anaesth.* 1954;26(5):323-32.

Endotracheal Tubes, Lung Isolation Devices and Combitube

Atul Prabhakar Kulkarni, Aparna Chatterjee, Swapnil Parab

Q. What is an endotracheal tube (ETT)?

Ans. An ETT is a device used to secure a patient's airway. It can be inserted via the oral or nasal route and is shaped accordingly.

Q. What is the material of the ETT? How is it standardized?

Ans. The ideal material should have following characteristics:

- Low cost
- Nontoxic and nonirritant to tissues
- Transparent so that exhaled air mist can be seen through it
- In case of nondisposable tubes should be easily sterilized and repeated sterilizations should not affect their durability
- Nonflammable
- Inside and outside surface of the tube should be smooth and nonwetable to prevent secretion buildup, allow easy passage of suction catheter or bronchoscope and prevent trauma to oral/nasal structures during passage
- Thin-walled with sufficient strength prevent occlusion of lumen from extrinsic compression and kinking
- Thermoplasticity to conform patients anatomy when in place
- Nonreactivity with lubricants and anesthetic agents
- Latex free.

Endotracheal tubes are made of red rubber or polyvinyl chloride (PVC). Other materials such as silicone rubber (polydimethylsiloxane) or Teflon have also been used.

Red Rubber ETT

Advantage:

- Easily cleaned, sterilized and reused.

Disadvantages:

- Becomes floppy and sticky with repeated use
- They are not transparent, hence secretions, blood cannot be seen
- Can easily kink and get clogged by secretions
- They do not soften at body temperature

- Risk of latex allergy (red rubber tubes are coated with latex)
- Thicker wall (in comparison to a PVC tube of the same size).

Polyvinyl Chloride

The routinely used ETTs.

Advantages:

- Disposable, inexpensive and have a lower tendency to kink as compared with red rubber tubes
- Nonirritant, compatible with tissues and cause less tissue reaction particularly with long-term intubation
- Stiffer, therefore facilitates intubation but soften at body temperature and conforms to patient's upper airway, reducing pressure at the point of contact
- Smooth nonwetable surface facilitates easy passage of a suction catheter or a bronchoscope
- Transparent
- *Thin wall:* Therefore, a wider lumen and thus a reduction in airway resistance for the same size tube as compared to a red rubber ETT.

Disadvantage:

- Cannot be used in laser surgeries, as PVC tubes can ignite and cause airway fires.

Tissue Reactivity of ETT

The material used for construction of the ETTs is tested to ensure that it is nonirritant. It must pass the American Society for Testing and Materials (ASTM) Standards or the American National Standards Institute (ANSI) testing. Tissue implantation test is carried out. The material is implanted in the paravertebral muscle of anesthetized rabbits under sterile conditions and the sites observed for any sign of inflammation. Tubes which pass the test have F-29, Z-79 or ASTM written on them. A CE mark on the tube also indicates compliance with the requirements of the Medical Devices Directive.

Q. Describe the design of the standard PVC ETT. What are the markings on the ETT?

Ans.

Design of a PVC ETT

It is a transparent tube with a preformed curve and made of PVC. It has two ends: a patient end (enters patient's trachea) and a machine end (which gets attached to the breathing circuit). The tube may be shortened at this end if required. A typical ETT is shaped like an arc of a circle with a radius of curvature of 140 ± 20 mm.

The patient end is cut obliquely and is called the bevel. The angle that the bevel makes with the longitudinal axis of the tube is called the angle of the bevel and it is about $38 \pm 10^\circ$. The bevel faces to the left and facilitates the visualization of the cords as the tube is being inserted from the right side of the mouth. On the opposite side of the bevel is a hole in the wall of the tube, called a Murphy's eye. It provides an alternate pathway for gas flow should the bevel get occluded. The tracheal tube standard specifies that the area of a Murphy's eye must not be $<80\%$ of the cross-sectional area of the tube lumen.

Tracheal tubes, which lack the Murphy's eye, are known as Magill-tipped ETTs (PVC tubes). The tip of the tube is smooth and atraumatic. As there is no Murphy's eye, the cuff is placed closer to the tip of the tube, decreasing the risk of inadvertent endobronchial intubation (Fig. 1).

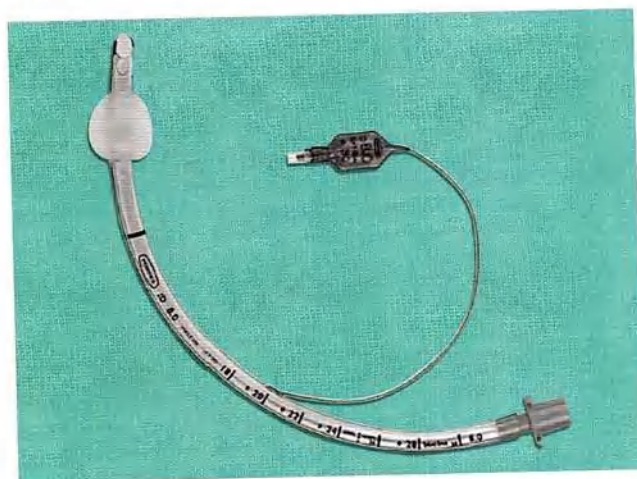


Fig. 1: Polyvinyl chloride endotracheal tube.

Markings on the ETT

There is a radiopaque line along the entire length of the tube which helps to determine the correct position of the tube on an X-ray if required. The length of the tube has transverse black markings at each centimeter along the outer curvature, which indicates the distance from the tip of the tube. A black transverse depth marker, which assists proper placement of the tube, is present on certain tubes

approximately 3 cm proximal to the cuff in size 6 and above (Note that this distance will vary according to manufacture).

For pediatric cuffed tubes, the depth marking from tip of tube varies according to size as given in Table 1.

TABLE 1: Endotracheal tube (ETT) marking in pediatric ETTs.

Internal diameter (mm)	Distance of depth marking from tube tip (mm)	Cuff-free subglottic tube length (mm)
3	24	9
3.5	27	10
4	30	12
4.5	34	12
5	39	16
5.5	45	16

The tubes are also marked with their internal diameter (ID) and outer diameter in millimeter, whether for oral or nasal use (red rubber tube) and the implantation test number. Note such as "for single use only" also may be written on the tube. ETTs have a uniform wall thickness. The machine end is circular and is fitted with a standard 15 mm universal connector which is compatible with standard circuits.

Q. Describe the cuff system of the ETT and types of the cuff.

Ans.

Cuff System of the ETT

Cuff system of an ETT consists of:

1. **Cuff:** The cuff is an inflatable sleeve near the patient end of the tube. Confirmed ASTM/ISO standards—specify the maximum distance of the cuff from the tip of tube (varies with tube size as shown in Table 1). Bonded edge of the cuff does not encroach on the Murphy's eye.
2. **Inflating system:** It includes an internal (built in the wall of the tube) inflation tube, an external inflation tube, a pilot balloon (located near the midpoint of the inflation tube or adjacent to the inflation valve), and a one-way valve (Cuff on red rubber tubes has a cap and not a one-way valve). The ASTM/ISO standard for external inflation tube requires that its external diameter does not exceed 2.5 mm and it to be attached to the tube at a small angle. The cuff material should be thin, soft but strong and resistant to tearing.

Advantages of a Cuffed Tube

- It prevents aspiration by sealing off the trachea from the oral cavity and gastric contents
- It avoids gastric distention during intermittent positive pressure ventilation (IPPV)
- It allows efficient ventilation.

Overinflation of the cuff leads to a high pressure within the cuff which gets transmitted to the tracheal mucosa. The pressure on the lateral tracheal wall measured at end expiration should be approximately 20–30 cm of H_2O (15–22 mm Hg) in a normotensive adult. This could worsen during the periods of systemic hypotension, thus overfilling of the cuff is not recommended.

The increase in pressure depends on the cuff compliance. To avoid this problem:

- The cuff can be filled with either the gas mixture identical to that used for the anesthesia or with sterile saline.
- The cuff should be inflated very gradually till the leak just disappears.
- The intracuff pressure should be monitored at frequent intervals during anesthesia.

After prolonged intubation as in an intensive care setting, there can be mucosal sloughing and ulceration at the point of contact of the cuff with the tracheal mucosa and intracuff pressures should therefore be monitored regularly.

Nitrous oxide (N_2O) can diffuse into the cuff filled with air during an anesthetic and with time increase the volume of the cuff and the pressure exerted on the tracheal mucosa. The rate of diffusion depends on:

- The permeability of the cuff material
- The surface area of cuff exposed to N_2O
- The partial pressure of N_2O .

Types of Cuff

High-volume low-pressure cuff: As in PVC tubes. This cuff has a large resting volume and area of contact with the tracheal wall hence, the pressure is distributed over a large area rather than at a specific point (**Fig. 2**).

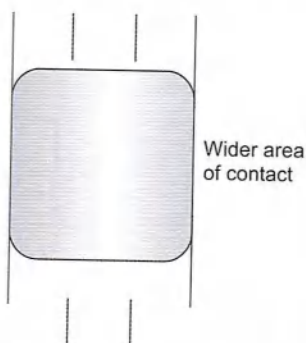


Fig. 2: High-volume, low-pressure cuff.

Advantage:

- Exert minimal pressure on the tracheal wall.

Disadvantages:

- Cuffs are made of a thin material and could be torn/damaged during intubation especially during a nasal intubation or if held with a Magill's forceps.

- The cuff may not be fully unfolded when seal is achieved; leaving some folds that may allow aspiration.

Low-volume, high-pressure cuff: As in red rubber or flexometallic/reinforced tubes (**Fig. 3**).

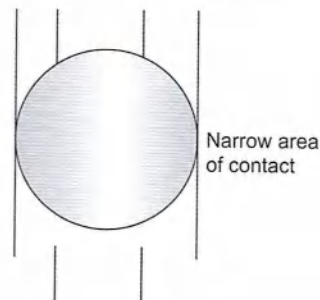


Fig. 3: Low-volume, high-pressure cuff.

Advantage:

- It gives better protection against aspiration of secretions into the lungs.

Disadvantage:

- Exert a high pressure on the tracheal wall and if left in situ for a prolonged period may lead to necrosis of the tracheal mucosa.

Foam-filled cuffs: They are soft, low-pressure cuffs. Self-inflating polyurethane foam material replaces the air that is used to inflate the cuff. The cuff is actively deflated so as to collapse the foam prior to insertion. After intubation, the pilot tube is opened to atmosphere which allows the foam to expand and fill the cuff. Since, the pilot tube is left open, diffusion of N_2O does not further increase the cuff pressure.

Pilot balloon is situated near the inflating valve and gives an idea of the state of inflation and deflation of the cuff, i.e., acts as a pressure volume indicator.

Inflation valve is designed to fit a nozzle of the standard syringe. It is a one-way valve which prevents the escape of air once inflated.

Q. Enumerate the indications and contraindications of tracheal intubation.

Ans.

Indications

- **For surgery:** Trachea is intubated to administer general anesthesia:
 - To secure and maintain a free airway
 - To protect against aspiration of gastric/oral contents
 - To provide positive pressure ventilation
- **Nonsurgical conditions:** As a life-saving measure:
 - Cardiopulmonary resuscitation
 - In an unconscious or semiconscious patient who is unable to protect his/her own airway, for example, drug overdose, poisoning, central nervous system

(CNS) disease, head injury, polytrauma and patient with impaired laryngeal reflexes

- When patient's own respiratory attempts/drive is inadequate and needs mechanical ventilation
- Respiratory obstruction
- Tracheobronchial toileting in severe sputum retention.

Contraindications

These are no contraindications to intubation. Intubation in certain scenarios can be tricky.

Appropriate precautions should be taken when dealing with such patients. The difficult scenarios include:

- *Severe airway trauma* with distortion of upper airway, edema and bleeding makes endotracheal intubation difficult or impossible. Alternate mode of securing the airway is needed.
- *Laryngeal edema and epiglottitis*: Intubation with a smaller tube can be life-saving.
- *Aneurysm of the arch of aorta*: Long-standing aneurysm can erode into the tracheal wall and during intubation it can burst causing exsanguination.
- *Cervical spine injury*: There is a possibility of converting an incomplete cervical spine fracture into a complete one with resultant spinal cord damage. Intubation can be achieved with manual in-line stabilization on a spine board with absolutely no neck movement.
- *Traumatic laryngeal fracture*.

Q. Describe the procedure of endotracheal intubation.

Ans.

- Confirm starvation in elective circumstances (In case of an emergency, i.e., a patient with a full stomach, a rapid sequence induction with Sellick's maneuver is carried out)
- Assess the airway to identify the ease of intubation
- Start essential monitoring like electrocardiography (ECG), noninvasive blood pressure (NIBP) and pulse oximetry
- Secure an intravenous access
- Things kept ready prior to induction: SALT (Suction, Airways, Laryngoscope, ETT):
 - A functioning suction apparatus with an attached catheter, oropharyngeal and nasopharyngeal airways to overcome upper airway obstruction if required, at least two working laryngoscopes with proper sized blades, proper sized ETTs and a syringe for cuff inflation or cuff inflation device.
- *Position of the patient*: The aim is to position the oral, pharyngeal and laryngeal axes in one line, i.e., in the line of vision of the anesthetist to optimize vocal cord visualization. This can be achieved by: (1) Flexion at

the neck by keeping a head ring or thin pillow below the head of the patient to raise it by approximately 10 cm. (2) Extension at the atlanto-occipital joint. This position is called as sniffing the morning air position or the Chevalier Jackson position.

- Intravenous or inhalational induction of anesthesia till patient loses consciousness.
- Administer muscle relaxant to facilitate tracheal intubation after checking adequate of mask ventilation.
- Open the mouth with the right hand and introduce the laryngoscope into the right side of the mouth, using the left hand moving the tongue to the left. The blade (curved Macintosh) is passed over the surface of the tongue till it reaches the space between the base of the tongue and the epiglottis. Lift the laryngoscope in the direction of the handle without applying any pressure on the upper incisors. The glottis will be visible under the epiglottis as the epiglottis is lifted forward. If a straight blade (e.g., Magill's) is used, the tip is placed over the epiglottis and lifted as above. Could result in bradycardia as the dorsal surface of the epiglottis which is innervated by the superior laryngeal branch of the vagus is touched.
- After laryngoscopy, the proper size tube is passed from the right corner of the mouth through the vocal cords till the proximal edge of the cuff is beyond the cords or the transverse black line (if present on the tube) is at the cords. Confirm the intratracheal placement of ETT by auscultation and capnometry.
- The cuff is inflated till any leak of air disappears.

Q. How would you select the appropriate size of the tube for your patient?

Ans. A size 7.5 or 8 mm ID is chosen for an adult male and a size 7 or 7.5 mm ID is chosen for an adult female patient. However, there is evidence to suggest that the incidence of hoarseness and sore throat is increased with the use of tubes with ID > 7 mm. Therefore, currently there is a trend toward using smaller tubes even in adult patients. For nasal intubation, size of tube will be decided by size of external nares.

Pediatric ETT sizes are calculated based on the weight and age of the patient. The narrowest portion of a child's airway is the cricoid cartilage and circular, a correct size uncuffed tube will achieve a good fit with minimal air leak.

Uncuffed tubes had been in use in pediatric patients until 8–10 years of age till recently however with development of thin polyurethane cuffs, cuffed ETTs are available in all pediatric sizes. A commonly used formula for uncuffed tube selection in patients over 1 year of age is:

- Age in years/4 + 4.5 mm
- The modified Cole's formula $[4 + (\text{age}/4)]$ for children aged 2 and older is also used.

- The formula for calculating a cuffed tube size is: $\text{Age}/4 + 3 \text{ mm}$ (Khine's formula).

Microcuff ETTs developed for use in children consist of a short, ultrathin polyurethane cuff located away from the subglottic area (the narrowest part of the glottis in children and hence vulnerable to cuff induced damage). The cuff effectively seals the tracheal wall at pressures as low as 10 cm H₂O and fills the gap between the tube and the tracheal wall without folds. The Murphy's eye is absent (Table 2).

TABLE 2: Selection of endotracheal tube (ETT) size in pediatric population.

Age	Tube size ID in mm
10–14 years	7.0–7.5
7–10 years	6.5–7.0
4–6 years	5.5–6.5
1–4 years	4.5–5.5
6–12 months	4.5–5.0
1–6 months	4.0–4.5
Neonate	2.5–3.5
Premature	≤3

(ID: internal diameter).

A rough guide to estimate the size of the tube in case the age of the child is not known is to use a tube whose external diameter is the same as the distal phalanx of the little finger.

In pediatric age group: Length of the tube (cm) = $(\text{Age}/2) + 12 \text{ cm}$.

These are rough guidelines. One has to remember that PVC tubes being thin-walled usually one size larger tube will pass through the cords than the same external diameter red rubber tube. Since, the narrowest portion in children is at the cricoid cartilage, the tube that is able to pass through the cords may not always pass through the subglottic region and should never be forced through it as it may cause severe trauma to larynx. Oral intubation is preferred in children (below 11 years), as there is a risk of injury to the hypertrophied adenoids and profuse bleeding during a nasal intubation.

Q. How would you confirm the intratracheal placement of an ETT?

Ans. Following confirmatory criteria are used for intratracheal placement of an ETT:

- Visualize the tube passing through the vocal cords
- A capnograph/end-tidal carbon dioxide (EtCO₂) curve
- Feel and compliance of the reservoir bag
- Chest wall rise with inspiration
- Auscultation of chest wall
- Movement of the respiratory mist in the ETT during ventilation
- Absence of epigastric/gastric distention

- X-ray chest
- Fiberoptic confirmation
- Esophageal detector device
- *Sonomatic confirmation of the tracheal intubation (SCOTI)*: A lightweight battery-powered, sonomatic device which emits sound waves into the tube and analyzes the reflection
- Good patient color and pulse oximeter value.

Q. What are the other types of ETT which you know? Describe each in brief.

Ans.

Cole's Tube

It is used in neonates, designed for use in emergency neonatal resuscitation. Cole's tube is an uncuffed tube with a tapering patient end. It has a shoulder at the junction of the tracheal end and the broad proximal end. The shoulder prevents endobronchial intubation as well impacts in the larynx and provides an airtight seal. It was designed to minimize the resistance to flow; however, the resistance is paradoxically increased due to turbulence at the junction with the narrow portion of the tube. Cole's tube is sized according to the ID of the tracheal end. Can cause trauma to the larynx and trachea if, the shoulder is forced into the larynx. These tubes cannot be used for nasal intubation (Fig. 4).

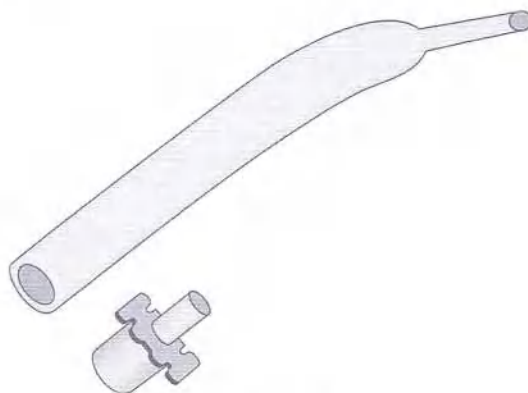
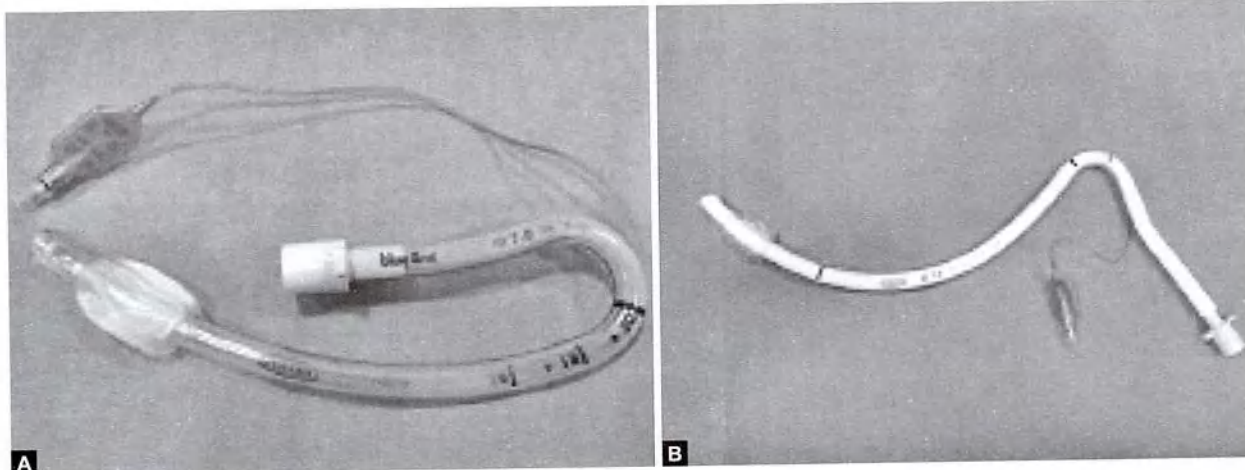


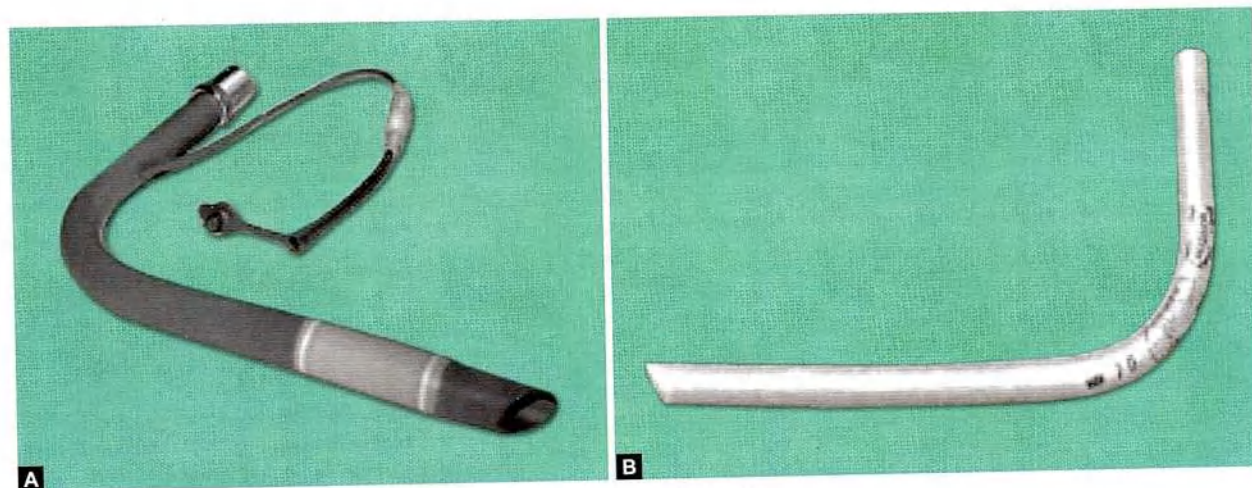
Fig. 4: Cole's tube.

Preformed Tubes (RAE or Ring-Adair-Elwyn Tubes)

These tubes have a preformed bend in the tube, so as to facilitate surgery on the head and face. They are made of PVC material. The external portion of the oral version (Fig. 5A—South polar) is bent at an acute angle so that it rests on patient's chin with the connector on chest away from operating field. It is used in cleft lip/palate/nasal surgeries. The nasal version (Fig. 5B—North polar) has a cephalic curve so that the connector rests on forehead. It is used for surgeries on the lower face, mandible, and floor of mouth.



Figs. 5A and B: Ring-Adair-Elwyn (RAE) tube.



Figs. 6A and B: Oxford tube.

The nasal and oral versions are available in various sizes both cuffed and uncuffed. There is a black mark at the bend, in most of the cases when it is positioned at the level of teeth or external nares in case of nasal tube, the tube is properly placed in the trachea. Selection of a tube of proper size is mandatory as the length of the tube beyond the bend is fixed. A smaller size tube may not comfortably reach below the glottis. While selecting the tube patients height and weight may be more useful than age.

Disadvantages: Tracheal suction is difficult due to bend in the tube; however, in an emergency, tube can be cut at the bend for proper suctioning. Selection of proper sized tube is essential for accurate positioning. The tube offers more resistance than a conventional tube.

RAE-flex tubes are also available which have conventional distal segment and spiral embedded proximal segment.

Oxford Tube

It is an L-shaped tracheal tube made up of red rubber. It may be cuffed or uncuffed. It is used for upper face surgeries as it is nonkinkable. The ID is uniform throughout the tube but the thickness of tube wall varies. The proximal portion is thicker than the tracheal segment thus allowing bigger tube to be passed through the trachea and the thicker wall at the lips prevents compression by the mouth gag. The bevel is situated posteriorly and may abut against tracheal wall if the head is flexed. The distance from the bevel to the curve is fixed hence a proper sized tube must be used to avoid endobronchial intubation. The tube offers greater resistance, difficult to suction through and is not transparent (**Figs. 6A and B**).

Flexometallic/Spiral Embedded/Reinforced/Armored ETTs

These are made of silicone rubber or PVC. Have a metal or nylon spiral within the wall of the tube which makes them

flexible and nonkinking. The spiral does not extend into the proximal and distal ends. It is easy to pass this tube over the fiberoptic bronchoscope (Fig. 7).

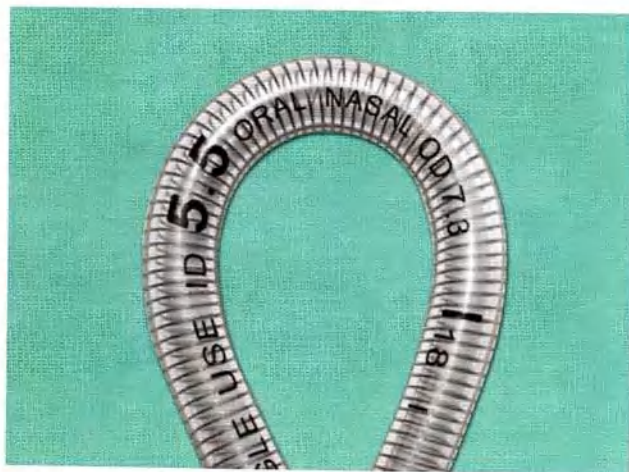


Fig. 7: Armored endotracheal tubes.

Disadvantages:

- **Thicker wall:** Hence, a smaller ID for a given outer diameter.
- **Difficult to insert:** A forceps or a stylet is required for intubation; tube may rotate on the stylet during insertion.
- Due to the spirals, it is not possible to cut the tube to the desired length. Risk of endobronchial intubation if not well-secured.
- The elastic recoil force may increase the incidence of accidental extubation hence the tube needs to be secured properly in place and a throat pack packed around the tube.
- On repeated use, the tubes become sticky and soft especially at the junction of the start of the spiral and the connector and liable to kinking at this spot.
- **The tube does not have a Murphy's eye:** It may result in obstruction if the bevel abuts against the tracheal wall.

Specific indications for these tubes are head, face and neck surgeries where the head and/or the neck is rotated or flexed. For example, neurosurgery, thyroidectomy, airway surgery like tracheal resection where the armored tube is passed through the tracheostomy stoma, spine surgery (prone position), etc.

Microlaryngeal Tracheal Surgery Tubes

It has a smaller external diameter and an ID of approximately 5 mm with an adult high-volume, low-pressure cuff (Cuff diameter is the same as on a standard 8 mm ID tube). This large cuff helps the tube to be in the center of the trachea. Some microlaryngeal tracheal surgery tubes (MLTs) may have a yellow-colored cuff for better visibility. It is designed

for microlaryngeal surgeries allowing better visibility and access to the surgical field. The tube offers a high resistance to gas flows and requires ventilation to be controlled with a long expiratory phase to prevent incomplete exhalation. The tubes are not laser resistant (Fig. 8).

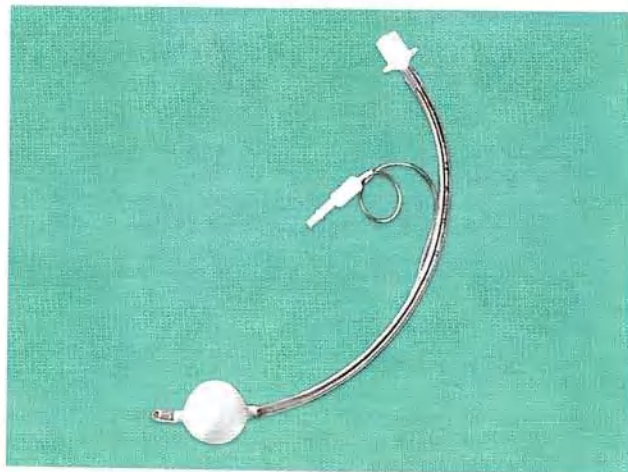


Fig. 8: Microlaryngeal tube.

Q. What are the complications of endotracheal intubation?

Ans. Complications of endotracheal intubation include:

- At intubation:
 - Trauma to lips, teeth, tonsillar pillar, tongue and nose, neck and jaw
 - Nasal, retropharyngeal, pharyngeal, uvular, laryngeal, tracheal, esophageal and bronchial trauma
 - Spinal cord and vertebral column injury
 - Vocal cord avulsions, fractures and dislocation of arytenoids
 - Hypertension, tachycardia, bradycardia and arrhythmias
 - Raised intracranial and intraocular tension
 - Laryngospasm, bronchospasm, laryngeal trauma
 - Airway perforation
 - Aspiration of gastric contents
 - Failed intubation
 - Esophageal intubation
 - Bacteremia especially with nasal intubation.
- While the ETT is in place:
 - Disconnection and dislodgment
 - Endobronchial intubation
 - **Airway obstruction:** By blood, secretions, foreign body, compression by a mouth gag, kinking
 - **Tension pneumothorax:** Due to overinflation of the lungs in a patient with an emphysematous bulla
 - Tracheal tube fire as in laser surgeries
 - Unsatisfactory cuff seal

- At extubation:
 - Cuff-related problems
 - ETT sutured to trachea or bronchus leading to difficult extubation
 - Laryngeal edema
 - Aspiration of oral or gastric contents.
- After extubation:
 - Sore throat, hoarseness
 - Laryngeal edema
 - Nerve injury
 - Superficial laryngeal ulcers
 - Laryngeal granuloma
 - Glottic and subglottic granulation tissue
 - Laryngeal synechiae
 - Vocal cord paralysis and aspiration
 - Tracheal stenosis
 - Sinusitis in prolonged nasal intubation.

Q. What is a double-lumen tube (DLT) and why it is used?

Ans. A DLT is essentially two single-lumen tubes of different lengths bound together and termed right- or left-sided depending on which main stem bronchus the tube is designed to fit. The shorter tube terminates above the carina and the longer tube (bronchial stem) extends into the main bronchus which it is designed to fit. The tube has two curves: (1) an anterior curve to fit the oropharyngeal-laryngotracheal curvature and (2) a second curve (bronchial curve) to the right or left depending on the bronchus that it fits into. The tube has two cuffs, one which seals the trachea and the other on the bronchial stem which seals the main bronchus. Both cuffs have separate inflation tubes, pilot balloons and connectors which are color coded for identification. The connectors are attached to the Y-shaped catheter mount and then attached to the breathing system. DLTs are available in right- and left-sided versions. The right-sided version has an eye in the bronchial cuff to allow ventilation of the right upper lobe. There is no eye in the left-sided DLT. The right-sided DLT is intended for the surgery on the left lung where the left lung needs to be collapsed with continued ventilation of the right lung. The left-sided DLT is intended for the use during the surgery on either right or left lung. Left-sided tubes are preferred even for left-sided surgery because of risk of occlusion of the right upper lobe bronchus with the bronchial cuff of a right-sided DLT.

Conditions where a right-sided DLT is absolutely indicated are:

- Left pneumonectomy
- Tumor in the left main stem bronchus
- Left lung transplantation
- Left bronchial trauma or distorted anatomy
- Left bronchial stent in place (which could be displaced if a left DLT is inserted).

Size: Conventional PVC DLTs were previously available in only adult sizes (35, 37, 39, and 41 Fr), but now smaller sizes are available. The smallest cuffed DLT is 26 Fr (Rusch) may be used in children as young as 8 years of age. DLTs are also available in sizes 28 and 32 Fr (Mallinckrodt Medical), suitable for children 10 years of age and older.

Marraro has described a bilumen tube for infants. This tube consists of two separate uncuffed tracheal tubes of different lengths attached longitudinally.

Q. What are the types of DLT?

Ans.

Types of DLT

Carlens tube (Fig. 9): Left-sided version with a carinal hook. One lumen anterior to the other. The carinal hook can traumatize the larynx or carina and make it difficult to insert. This tube may be especially useful with massive hemoptysis when verification of the tube placement is difficult. Lumen is small, hence greater resistance and difficult to suction down the tubes.



Fig. 9: Carlens tube.

White tube: A right-sided version similar to Carlens tube. It has a carinal hook. The cuff for the right main stem bronchus is circumferential superior to the opening to the upper lobe bronchus and continues distally behind the opening. Both Carlens and White tubes are not in clinical use due to their shortcomings and availability of better tubes in market.

Robertshaw tube: Made of red rubber. Both left and right versions are available. Lumens are side by side and wider than older tubes. Used for multiple use.

PVC tubes: Available from many companies.

Polyvinyl chloride DLT (Figs. 10 and 11): It is made up from tissue implantable plastic. The two D-shaped lumen lie side by side with a bronchial lumen at an angle of 20° in right side DLTs and 40° in left side DLTs. The lumens are wider

and hence offer less resistance to ventilation and allow suctioning. It has three ends and two curves, the tracheal end which lies in trachea and ventilates the lung being operated, the bronchial end lies in the main stem bronchus and ventilates the nonoperative lung and the machine end which is bifurcated to connect to the double catheter mount. The two curves are placed right angles to each other, the proximal corresponding to the oropharyngeal curve and the distal corresponding to the bronchial curve as the main stem bronchus is given off from the carina. There are two cuffs, bronchial (blue-colored for easy identification through fiberoptic bronchoscope and tracheal (White) cuff, seal the respiratory tract from the gastrointestinal tract (GIT) and atmosphere. In right-sided DLTs, the bronchial cuff has a slot in its lateral aspect for upper bronchus ventilation. The carinal hook is absent. Malleable intubation stylet and suction catheters are packaged with the tube.

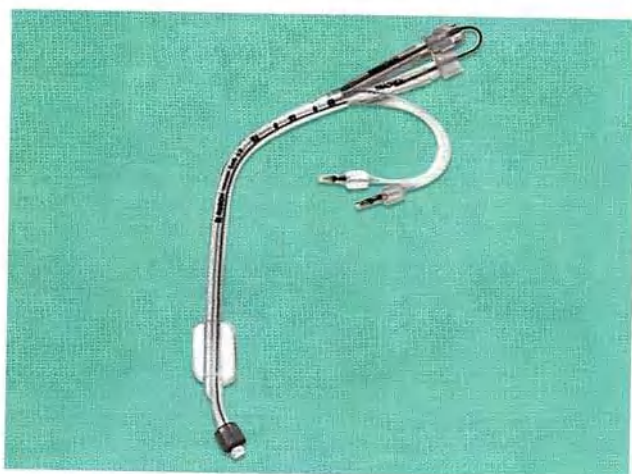


Fig. 10: Double-lumen tube.

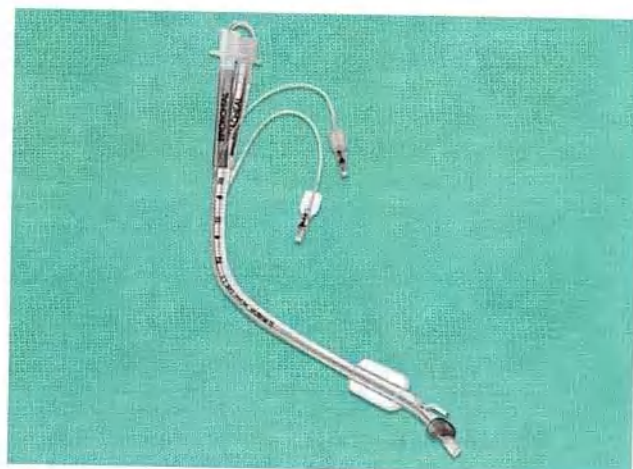


Fig. 11: White double-lumen tube.

They are available in five sizes—41, 39, 37, 35, and 28 corresponding to IDs of 6.5, 6.0, 5.5, 5.0, and 4.5 mm,

respectively. They are larger in size as compared to any other DLT of same size and for single use only.

DLT with in-built camera (VivaSight-DL): This is the new version of conventional DLT, in which a built-in camera is located at the opening of tracheal lumen of the tube (Fig. 12). This camera helps in insertion and confirmation of the DLT. It also allows to monitor the position of DLT continuously throughout the surgery. It also has the provision of flushing and cleaning the camera tip if the vision becomes blurred due to secretions of blood. With the use of this DLT, the requirement of pediatric bronchoscope is minimized.

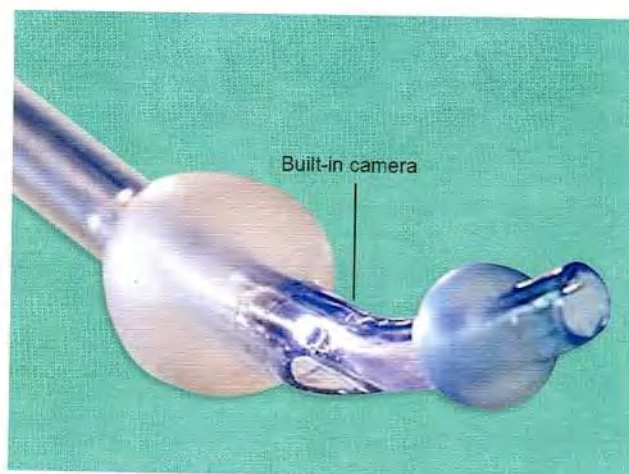


Fig. 12: Distal end of the VivaSight-DL.

Q. How is the right main stem bronchus different to the left and how does this affect right-sided DLT design?

Ans. The adult trachea begins at the lower end of the cricoid cartilage (C6 vertebral level) and bifurcates behind the manubriosternal joint (T4). It is 15 cm long. The main differences between the right and left main bronchi include the following:

- Right bronchus is shorter wider (supplies larger lung) and diverges away from the trachea at a 25° angle, while the left bronchus diverges at a 45° angle.
- Left bronchus divides into only upper and lower lobe branches while the right bronchus has upper, middle, and lower lobe branches.
- It is difficult to correctly position a right-sided DLT because of anatomic variations between individuals in the distance between the right upper lobe orifice and the carina often resulting in obstruction of right upper lobe bronchus and difficulty in ventilating the right upper lobe. The orifice of the right upper lobe bronchus from the carina is 15 mm in women and 20 mm in men, while that of the left upper lobe is 50 mm from the carina. Right-sided DLTs therefore have

a slit in the bronchial cuff for ventilating the right upper lobe. Correct placement requires confirmation with a fiberoptic bronchoscope (**Fig. 13**).

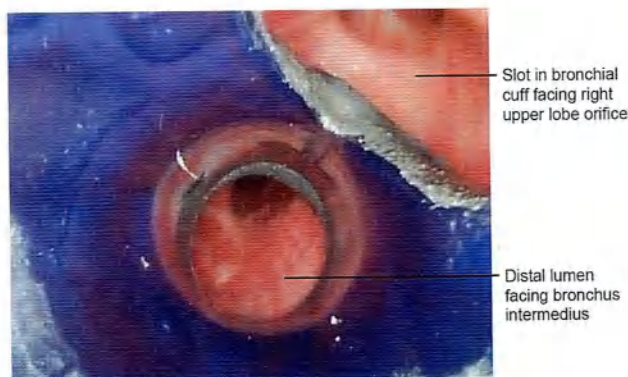


Fig. 13: Bronchoscopic confirmation of right double-lumen tube (DLT) position.

Choosing the appropriate size double-lumen ETT and insertion technique: See chapter on "Pneumonectomy".

Q. What are the complications of DLTs?

Ans. Complications include:

- Upper airway trauma during insertion
- Malpositioning resulting in hypoxia, inability to ventilate good lung, inability to deflate operative lung, etc.
- Disruption of tracheobronchial tree
- Suturing the endobronchial cuff or tube to the bronchial stump resulting in difficult extubation
- Traumatic laryngitis.

Q. How do you confirm the correct positioning of DLT?

Ans. The confirmation of DLT position is performed using a pediatric fiberoptic bronchoscope (external diameter 3.5 mm). Upon passing the bronchoscope through the tracheal lumen, one should be able to see an empty bronchus and other bronchus carrying the bronchial segment of the DLT. For example, in a correctly placed left-sided DLT, view through tracheal lumen should show unoccupied right main bronchus and left main bronchus harboring the bronchial segment of DLT (**Fig. 14**). Bronchoscope should be passed through the empty bronchus for correct identification of bronchus. Right main bronchus has an opening for right upper lobe, which makes its identification easy. Also, one should not see the blue bronchial cuff above the carina. Afterward, the bronchoscope is passed through the bronchial lumen and position of the tip of bronchial segment is confirmed to be above secondary carina. Bronchoscopy is considered as the gold standard for confirmation of position of DLT. It ensures anatomical separation of the two lungs.

The distribution of the regional ventilation is confirmed by auscultation. Recently, lung ultrasound has been shown

to be more accurate in confirming regional distribution of ventilation than auscultation.

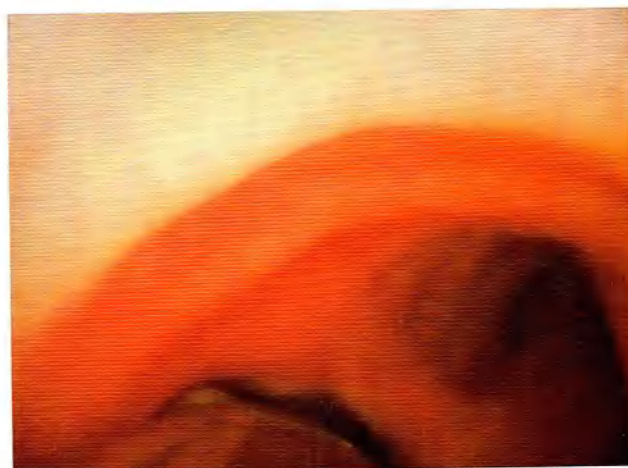


Fig. 14: Bronchoscopic view through tracheal lumen of left double-lumen tube (DLT). Right bronchus is unoccupied while rim of bronchial cuff is seen beyond the carina on left side.

Q. What is a bronchial blocker (BB)? What are various types of BBs?

Ans. In contemporary thoracic anesthesia, there are five BBs that have been introduced in clinical practice in recent years: (1) the Arndt blocker, (2) the Cohen flexitip blocker, (3) the Fuji Uniblocker, (4) the Coopdech endobronchial blocker tube, and (5) the EZ-blocker.

The basic principle of successful lung isolation with a BB relies on familiarity with the devices, knowledge of tracheobronchial anatomy, skills with fiberoptic bronchoscopy, and awareness of potential problems and complications.

Arndt blocker: The Arndt BB is available in 5 Fr, 7 Fr, or 9 Fr sizes. Lengths of the catheter are 65 cm and 78 cm with an inner lumen that measures 1.4 mm in diameter. Near the distal end of the catheter, there are side holes to facilitate lung deflation. These side holes are present in the 9 Fr Arndt blocker only. The Arndt blocker has a high-volume, low-pressure cuff with either an elliptical or spherical shape. A unique feature of the Arndt blocker is that the inner lumen contains a flexible nylon wire passing from the proximal to the distal end of the catheter, which exists as a small flexible wire loop. The wire loop of the Arndt blocker is coupled with the fiberoptic bronchoscope and serves as a guide to introduce the blocker into a bronchus (**Fig. 15**).

Cohen flexitip blocker: The Cohen blocker is available only in 9 Fr size and has 65 cm length with an inner lumen measuring 1.6 mm in diameter. This device comes with a spherically shaped balloon with a high-volume, low-pressure cuff. Near the distal end of the catheter there are

side holes incorporated to facilitate lung deflation. The Cohen blocker relies on a wheel-twisting device located in the most proximal part of the unit that allows deflecting of the tip of the distal part of the blocker into the desired bronchus. This device has been purposely preangled at the distal tip to facilitate insertion into a target bronchus. Also, there is a torque grip located at the 55 cm mark to allow rotation of the blocker. In the distal tip above the balloon, there is an arrow that when seen with the fiberoptic bronchoscope indicates which direction the tip deflects (Fig. 16).

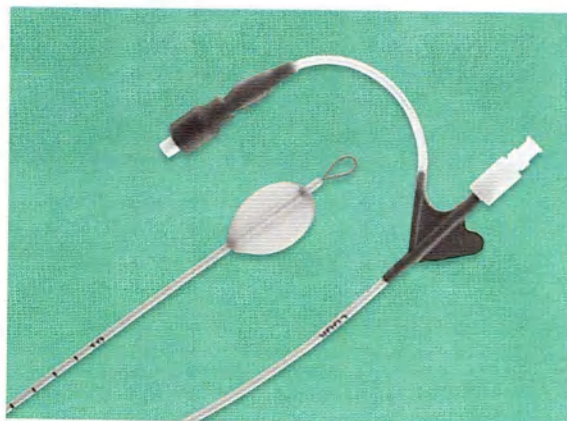


Fig. 15: Arndt bronchial blocker.

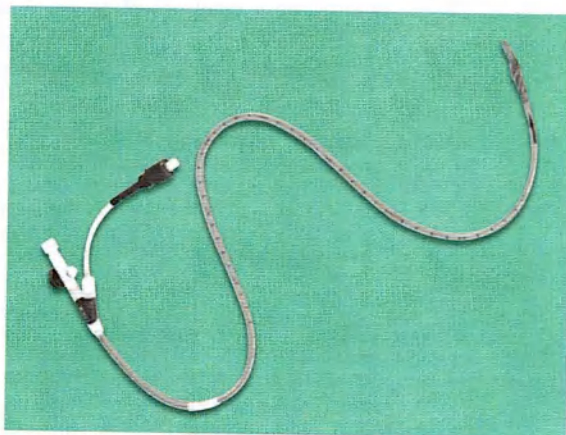


Fig. 16: Cohen flexitip blocker.

Fuji Uniblocker: The Fuji blocker is an independent BB that is available in 4.5 Fr and 9 Fr size and is 65 cm in length and has a high-volume balloon made of silicone with a gas barrier property to reduce diffusion of gas into or out of the cuff. The Fuji blocker has a preshaped tip that allows guidance through the desired bronchus (Fig. 17).

Coopdech blocker: Coopdech blocker is similar to Fuji Uniblocker in terms of preangled distal tip, and low-pressure barrel-shaped silicone cuff. It is available in 9 Fr size only. The central channel allows lung deflation,

application of continuous positive airway pressure (CPAP) and suction (Fig. 18).

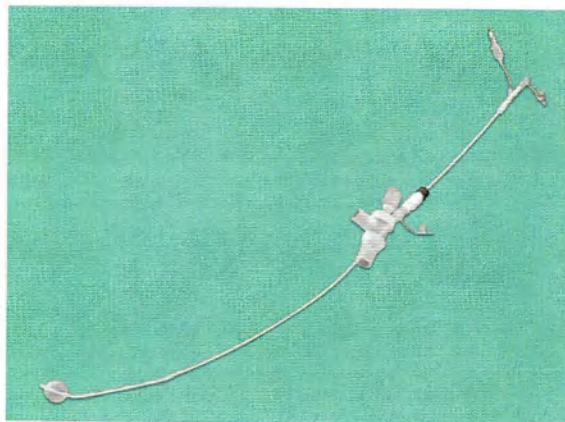


Fig. 17: Fuji uniblocker.

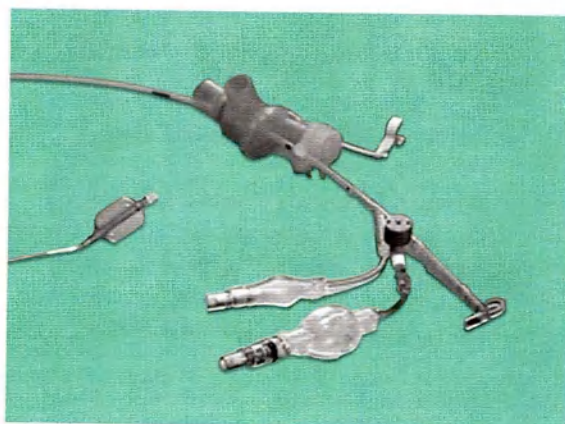


Fig. 18: Coopdech endobronchial blocker.

EZ-blocker: The EZ-blocker is a new BB that has a Y-shaped distal end. The peculiar characteristic of this device is that both distal ends are fitted with an inflatable cuff and a center hollow channel in each end. Therefore, each inflatable balloon can be placed on the right or left main stem bronchus, respectively. The shape of this balloon is spherical and there is only one size available at the present time, 7 Fr (Fig. 19).

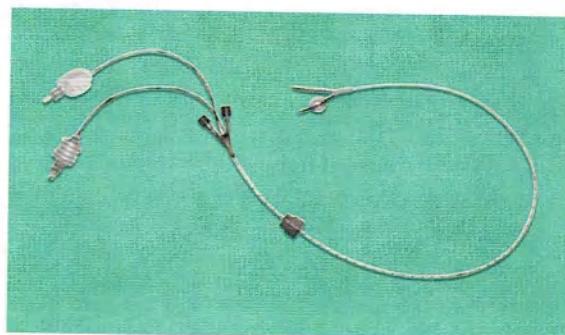


Fig. 19: EZ-blocker.

All independent BBs come with a multiport connector where a blocker and a fiberoptic bronchoscope can be passed separately.

Q. Describe the method of insertion and confirmation of BB.

Ans. The largest possible single-lumen ETT is preferred as it facilitates an easy passage and advancement of the blocker along with the flexible fiberoptic bronchoscope (3.5 or 4.0 mm outer diameter). Prior to the advancement of the BB, a complete fiberoptic bronchoscopy examination is recommended to ensure that the distal tip of the single-lumen ETT is approximately 2–3 cm above tracheal carina and also to inspect the bronchial anatomy. Lubricated BBs and the flexible fiberoptic bronchoscope are then advanced through the ETT. With the Cohen, Fuji Uniblocker, and EZ-blocker, one can observe the direction of the tip of the blocker and direct the blocker into a desired target bronchus. In contrast, with the Arndt blocker, the flexible bronchoscope is ahead of the tip of the blocker, hence tip of the blocker is not visible. Bronchoscope coupled with the blocker is advanced into desired bronchus. Once into the desired position, bronchoscope is decoupled from the blocker and is withdrawn under vision, keeping the blocker in situ.

All the blockers need to be advanced 1 or 2 cm beyond the tracheal carina into the bronchus while the patient is still in the supine position; optimal placement is achieved when the outer surface of the blocker balloon is seen at least 1 cm below the tracheal carina and a proper seal is obtained when the balloon is inflated with approximately 5–8 mL of air.

For a left-sided main stem bronchial blockade, turning the patient's head to the right and twisting the torque part of the blocker facilitates insertion into the left bronchus.

It is preferable to deflate the BB balloon before turning the patient to lateral decubitus position. Blockers with inflated balloon tend to get dislodged during positioning of the patient.

Q. How do we make the BB work?

Ans. In order to expedite lung collapse, following strategies are used:

- During two-lung ventilation, the lung to be deflated is ventilated with 100% oxygen or O₂ with N₂O mixture. O₂ and N₂O, being rapidly diffusible gases, help in quick deflation of the lung.
- *Disconnection technique:* Ventilator is disconnected from the tube, BB is deflated, and lung is allowed to collapse. Oxygenation during this time is monitored by pulse oximeter. After deflation of the lung, BB cuff is inflated under bronchoscopic vision and then ventilation is resumed. This method allows quick escape of the air trapped in the lung and hastens the process of lung collapse.

- In addition, suction should be applied through the central channel of the blocker to hasten the collapse of the lung.

Q. Discuss the advantages and disadvantages of DLTs and BBs.

Ans. The advantages and disadvantages of DLTs and BBs are summarized in **Table 3**.

Q. Which patients will benefit from the use of BBs?

Ans. In contemporary thoracic anesthesia, BB can be used for any patient who requires lung isolation and is scheduled for minimally invasive lung or cardiac surgery. In addition, BBs are particularly useful in following cases:

- Predicted difficult intubation
- Nasotracheal intubation
- Selective lobar blockade
- Any patient with an existing tracheostomy in place, or
- Patients in whom postoperative mechanical ventilation are anticipated.

TABLE 3: Advantages and disadvantages of double-lumen tubes and bronchial blockers.

Double-lumen tubes	Bronchial blockers
Advantages: • Large lumen facilitates suctioning or application of continuous positive airway pressure (CPAP) • Preferred for absolute lung separation • Conversion from two- to one-lung ventilation is easy and reliable • Less incidence of intraoperative dislodgment	Advantages: • Preferred in patients with difficult airway/tracheostomy • No need to change the tube if mechanical ventilation is required • Minimum chances of airway trauma • Preferred for selective lobar blockade
Disadvantages: • Higher incidence of airway trauma • May not be available in proper size especially in pediatric patients • Not preferred in difficult airways • Need to change to single-lumen tube if mechanical ventilation is required	Disadvantages: • Narrow central channel—difficulty in suctioning or application of CPAP • Higher chances of dislodgment intraoperatively

Q. What are the potential problems and limitations with the use of BBs?

Ans.

- One must be aware and alert of potential malposition and its catastrophic consequences. For example, dislodgment of the inflated BB into the trachea, results in occlusion of the tracheal lumen, and an inability to ventilate.
- With potential chances of dislodgment, BBs are not preferred in cases where chances of contamination from one bronchus to other are high like in cases of bronchial lavage or bleeding within bronchial tree.
- During pneumonectomy, BB needs to be withdrawn from the bronchus before application of the stapler

across the bronchus. This may temporarily loose the lung isolation.

- In case of endobronchial growth, passage of blocker into the bronchus can be hazardous as it may traumatize the tumor.

Q. What are the indications of one-lung anesthesia?

Ans.

Indications:

- Absolute:
 - Isolation of one lung from the other to avoid spillage or contamination:
 - Infection
 - Massive hemorrhage
 - Control of the distribution of ventilation:
 - Bronchopleural fistula
 - Bronchopleural cutaneous fistula
 - Surgical opening of a major conducting airway
 - Tracheobronchial tree disruption
 - Life-threatening hypoxemia due to unilateral lung disease
 - Unilateral bronchopulmonary lavage
 - Video-assisted thoracoscopic procedures
- Relative:
 - Surgical exposure (high priority):
 - Thoracic aortic aneurysm
 - Pneumonectomy
 - Upper lobectomy
 - Mediastinal exposure
 - Thoracoscopy
 - Surgical exposure (low priority):
 - Middle and lower lobectomies and subsegmental resections
 - Esophageal surgery
 - Thoracic spine procedure

- Minimally invasive cardiac surgery [minimally invasive direct coronary artery bypass grafting (MIDCABG)]

- Severe hypoxemia due to unilateral lung disease.

Q. Enumerate methods of isolation of lung other than DLT/BB.

Ans. It may not be possible to pass a DLT in all cases. For example, in children, distorted airway anatomy, nonavailability of the tube or appropriate size. In such cases, the isolation can be achieved by:

- Introducing a BB into the operative side to prevent blood and secretions trickling into the normal side and soiling it
- Introducing a Fogarty catheter or a Foley's catheter into the operated bronchus serving as the BB
- Univent tube
- Passing a single-lumen tracheal tube into the bronchus of the nonoperated lung (not a very good option).

Q. What is Combitube?

Ans. Combitube is a DLT that allows blind placement in either the trachea or the esophagus. It is a single use device. It is available in two adult sizes 37 Fr and 41 Fr only (**Fig. 20**). Combitube consists of:

- A large proximal (oropharyngeal) cuff, inflated with 100 mL of air. Seals the pharynx by filling the space between the base of tongue and soft palate so that no gas escapes through the mouth.
- A distal (esophageal/tracheal) cuff inflated with 15 mL of air.
- Two lumens, fused longitudinally with a universal connector attached to each end. One lumen opens beyond the distal cuff, while the other lumen (usually colored blue) ends between the two cuffs (blind end) and has eight ventilating ports located in between the two cuffs.

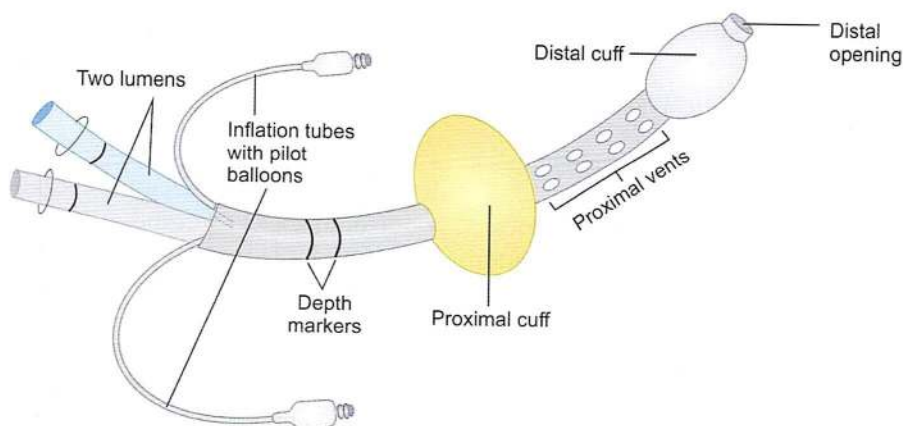


Fig. 20: Combitube.

- Two inflation tubes with a pilot balloon and a self-sealing valve each. Pilot balloon to proximal cuff is also blue colored.
- A distal opening beyond the distal cuff opens in the trachea or the esophagus depending on the tube's position.
- There are two depth marks on the airway tube.

This tube is used for emergency airway management and not for anesthesia. It has been included as the alternative airway device in the guidelines for advanced cardiac life support (ACLS) of the American Heart Association (AHA), practice guidelines by Difficult Airway of USA, the Canadian Airway Focus Group and also the European Resuscitation Group.

When inserted blindly, tube enters the esophagus in 95% cases. Ventilation is possible regardless of whether the tip is in the trachea or the esophagus and IPPV may be performed through either tube depending on the device's position.

The tube after blind insertion is advanced till the two black depth markers are at the level of the incisors. The proximal cuff is inflated first.

When the tip is in the esophagus, the distal cuff seals the esophagus and protects against aspiration. A gastric tube can then be placed through that lumen. The proximal cuff seals the pharynx by filling the space between the base of tongue and soft palate so that no gas escapes through the mouth. Always ventilate through the blue lumen first. The air escapes through the eight ventilating ports between the two cuffs. It cannot enter esophagus because of the seal formed by the inflated distal cuff. If ventilation through the blue lumen is not possible, then the distal tip is possibly in the trachea, ventilate through the other lumen.

If ventilation is not possible through either lumen, it may be because of obstruction of the larynx by the proximal balloon. The device should hence be withdrawn by 1-2 cm and ventilation attempted again.

The device has been in use in prehospital settings by paramedical personnel for over 20 years and has been used successfully in patients with difficult airway following severe facial burns, trauma, and upper airway bleeding and vomiting where it was not possible to visualize the vocal cords. However, its use is contraindicated in the following scenarios:

- Upper airway obstruction due to foreign body or tumor
- In case a patient has ingested some caustic agent.

Disadvantages:

- Increased incidence of a sore throat, dysphagia and upper airway hematoma
- Risk of esophageal rupture
- Not possible to suck out the secretions and/or blood from the pharynx.

Q. How would you sterilize the ETTs?

Ans. Disposable tubes are available at such a low cost that it is usually not cost-effective to use reusable tubes.

If the tube is reusable, secretions should be removed by rinsing and then soaking in a detergent solution. Remove the connector and rinse the tube thoroughly with soap water, making certain to flush the lumen and then dry the tube. This tube then can be sterilized by:

- *Steam sterilization (autoclaving):* Steam sterilization under pressure kills all bacteria, spores, and viruses. Repeated autoclaving makes them soft, more liable to kink and decreases the elasticity of the cuff.
- *Ethylene oxide (EtO):* EtO is a popular method for sterilizing tracheal tubes as it is method of choice for heat-sensitive items. However, with repeated gas sterilization, rubber tracheal tubes soften and kink more easily.
- *Liquid chemical disinfection:* It has been used for tracheal tubes. The inflation tube should be closed (capped) during immersion to prevent the solution from entering the cuff. The tube frequently floats and needs to be held submerged with an object. Thorough rinsing is necessary after use of all chemical agents except alcohol. Pseudomembranous laryngitis has been reported following disinfection of tracheal tubes with glutaraldehyde.

SUGGESTED READING

1. Al-Shaikh B, Stacey S. Essentials of Anaesthetic Equipment, 2nd edition. New York: Churchill Livingstone; 2001.
2. Brodsky JB, Macario A, Mark JB. Tracheal diameter predicts double-lumen tube size: a method for selecting left double-lumen tubes. *Anesth Analg*. 1996;82:861-4.
3. Campos JH. An update on bronchial blockers during lung separation techniques in adults. *Anesth Analg*. 2003;97:1266-74.
4. Davey A, Diba A. Ward's Anaesthetic Equipment, 5th edition. London: Saunders Elsevier; 2005.
5. Hammer GB. Pediatric thoracic anesthesia. *Anesthesiol Clin North Am*. 2002;20(1):153-80.
6. Hannallah M, Benumof JL, Silverman PM, Kelly LC, Lea D. Evaluation of an approach to choosing a left double-lumen tube based on chest computed tomographic scan measurement of left main stem bronchial diameter. *J Cardiothorac Vasc Anesth*. 1997;11:168-71.
7. Harrop-Griffiths W, Ellis H, Feldman S. Anatomy for Anaesthetists, 8th edition. Oxford: Blackwell Publishing; 2004.
8. Khine HH, Corddry DH, Ketrick RG, Martin TM, McCloskey JJ, Rose JB, et al. Comparison of cuffed and uncuffed endotracheal tubes in young children during general anesthesia. *Anesthesiology*. 1997;86(3):627-31.
9. Marraro G. Selective bronchial intubation in paediatrics: the Marraro pediatric bilumen tube. *Paediatr Anaesth*. 1994;4:255-8.
10. Seegobin RD, van Hasselt GL. Endotracheal cuff pressure and tracheal mucosal blood flow: endoscopic study of effects of four large volume cuffs. *Br Med J (Clin Res Ed)*. 1984;288(6422):965-8.

Cardiopulmonary Resuscitation

Sheila Nainan Myatra, Bhakti Trivedi, Amol Trimbakrao Kothekar

The 2015 American Heart Association (AHA) guidelines update for cardiopulmonary resuscitation (CPR) and emergency cardiovascular care have further refined the 2010 AHA guidelines for CPR, targeting improved survival from sudden cardiac arrest (SCA). These guidelines are based on the most extensive review of literature of CPR and specially designed to reduce the information the rescuer needs to remember and also simplify the important skills he needs to perform.

This update was published in Circulation in November 2015. The following article is based on these recommendations.

Q. What is cardiac arrest and how will you clinically diagnose it? What is CPR?

Ans. Cardiac arrest is the cessation of all cardiac mechanical activity. Its clinical diagnosis is confirmed by:

- Unresponsiveness
- Absence of detectable pulse
- Apnea or agonal respiration (gasp type of breathing).

Cardiopulmonary resuscitation is an attempt to restore spontaneous circulation through any of a broad range of maneuvers and techniques. If CPR is delayed or delivered inappropriately, the survivor may have various degrees of neurological dysfunction, which is undesirable. Thus, all attempts at CPR should be performed with the aim of return of spontaneous circulation (ROSC) with intact cerebral function. Thus, CPR has been rightly termed as cardiopulmonary cerebral resuscitation (CPCR).

Q. List the cardiac arrest rhythms, which of these are amenable to defibrillation.

Ans. The four cardiac arrest rhythms are:

1. Asystole
2. Pulseless electrical activity (PEA)
3. Ventricular fibrillation (VF)
4. Pulseless ventricular tachycardia (VT).

Ventricular fibrillation and pulseless VT are amenable to defibrillation.

Q. What is the management of a patient who has collapsed in your presence? Enumerate the sequence of basic life support (BLS) and advanced cardiac life support (ACLS).

Ans.

Step 1

Verify scene safety: Rescuers arriving on the scene of an emergency should ensure safe environment for the provider (person delivering CPR). A quick look at the patient's location and surroundings should be done to rule out imminent physical threats such as toxic or electrical hazards.

Step 2

Check response: Gently tap the patient on shoulders and check for a response. If victim is unresponsive, shout for nearby help and activate emergency response system. Ask for help with the defibrillator or if there is extra person send him to get the defibrillator.

Step 3

Look for no breathing or only gasping and simultaneously check for pulse for not > 10 seconds.

Remember short period of seizure-like activity or agonal gasps may occur in victims of cardiac arrest and often confuse the rescuer. Suspect cardiac arrest if there is no response and absent or agonal respiration.

The pulse check should be performed simultaneously with the check for breathing to minimize delay in detection of cardiac arrest and initiation of CPR. Pulse check if done using a central pulse (carotid or femoral) for no > 10 seconds. If pulse is not felt in 10 seconds or there is any doubt start chest compression.

Step 4

If there is no breathing or only gasping, with no pulse, start CPR immediately.

Initiate chest compressions before giving rescue breaths (A-B-C is now C-A-B).

Positioning:

- Victim should lie supine on a hard surface.
- Rescuer kneels beside the victim's thorax (either side).
- Keep arms straight, elbows locked and shoulder directly above the hand.
- Hand placement—place the heel of the hand on the lower half of the victim's sternum in the center (middle) of the chest, between the nipples, then place the heel of the second hand on top of the first so that the hands are overlapped and parallel. Interlock fingers to avoid compression on the ribs.

Technique: During CPR remember to “push hard and push fast”:

- **Compression rate:** 100–120/min.
- **Depth of sternal compression:** 5–6 cm [1/3rd of anteroposterior (AP) diameter in children and infants].
- **Compression-ventilation ratio:** Adult patient 30:2 (with one or more rescuers). Child or infant 30:2 (with one rescuer) and 15:2 (with more than one rescuer).
- **Compression-relaxation ratio:** 1:1 (allow complete recoil of the chest).
- Perform five cycles (approximately 2 minutes) of compression and ventilation (ratio 30:2).
- Switch the compressor every 2 minutes (i.e., after every five cycles).
- Give 2 minutes of uninterrupted CPR (limit interruptions to <10 seconds, interrupt only during intubation and just when you are ready to deliver a shock).

In adult cardiac arrest with an unprotected airway, chest compression fraction of at least 60% should be targeted. To easily achieve the above, one could use simple counting at the speed of approximately 100/min—“one and two and three and” Every time you say a number—compress and when you say “and” you relax, thus maintaining the 1:1 compression-ventilation ratio.

Airway and Ventilation

- Open the airway using a “head tilt-chin lift” maneuver (avoided when head and neck trauma is present or suspected) or “jaw thrust” maneuver.
- Give two slow rescue breaths using face mask and Ambu bag (using reservoir bag) to deliver 100% oxygen after every 30 compressions.
- Give one breath over 1 second (rapid ventilation which could cause gastric insufflations and increase the risk of aspiration).

- Give sufficient tidal volume to ensure visible chest rise.
- Reposition mask if there is a leak and insert an oral or nasal airway if there is airway obstruction due to tongue fall.
- Use of cricoid pressure during ventilation is generally not recommended.

Step 5

Attach defibrillator [automated external defibrillator (AED) or manual defibrillator] and shock if indicated:

- As soon as an AED/defibrillator is available irrespective of which step you have reached, attach it and shock if indicated, i.e., in VF and pulseless VT.
- Prefer a biphasic defibrillator, if unavailable use monophasic defibrillator.
- **Electrode placement**—anterior-lateral pad position (default). Alternative positions are anterior-posterior, anterior-left infrascapular and anterior-right infrascapular.
- **Shock energy—biphasic:** Use manufacturer recommendation (120–200 J), if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered if available. Monophasic: 360 J (In children and infants, use 2–4 J/kg first and 4 J/kg for subsequent shocks, higher energy may be considered but not to exceed 10 J/kg).
- CPR should be continued till defibrillator is ready for use.
Ensure that no one is touching the patient before you shock. Charge to the appropriate energy level, use the warning chant—“all clear”—then shock.
- No pulse check is recommended after defibrillation, resume CPR immediately.
- If nonshockable rhythm (asystole or PEA), continue chest compression.
- Reattach the defibrillator after every 2 minutes of CPR.
- Reduce time between the last compression and shock delivery and the time between shock delivery and resumption of compressions. CPR should be performed while the defibrillator is readied.
- There is no upper limit to the number of shocks you can give. Remember the shockable rhythms are the ones with the better prognosis so never give up. As long as VF/VT persists attempts at defibrillation must continue. As long as the myocardium has energy to produce VF, it has energy to produce a perfusing rhythm.
- AEDs can now be used even in infants with a pediatric dose attenuator, if manual defibrillator is not available. If neither is available AED without pediatric dose attenuator can be used (All AEDs are biphasic).

- The precordial thump may be considered in witnessed monitored unstable ventricular tachyarrhythmias only when a defibrillator is not available.
- Electric pacing is not recommended for routine use in cardiac arrest.

Step 6

Drug Therapy

- Use intravenous (IV) or intraosseous (IO) route for bolus delivery of drugs. For IV use, give bolus drug followed by a 20 mL saline push and raise the extremity (use special IO needles to deliver the drug; if using the IO route).
- If both IV and IO are unavailable then tracheal route, i.e., through endotracheal tube (ETT) may be used. Epinephrine, atropine, lidocaine, and naloxone may be administered through this route. Optimal ETT doses of medications are unknown, in general experts recommend use of 2–2.5 times the dose diluted 5–10 mL of distilled water or saline.
- Give a vasopressor soon after giving shock. Epinephrine IV/IO dose: 1 mg every 3–5 minutes. Pediatric dose of epinephrine 0.01 mg/kg (0.1 mL/kg 1:10,000) IV/IO and 0.1 mg/kg (0.1 mL/kg 1:1000) through ETT. Maximum dose is 1 mg IV/IO and 2.5 mg through ETT.
- Amiodarone should be given when VF/VT is unresponsive to CPR, defibrillation, and vasopressor therapy. IV/IO dose—first dose: 300 mg bolus and second dose: 150 mg (after 3–5 minutes if VF/VT recurs or persists). This may be followed by a 24-hour infusion (1 mg/min for 6 hours and then 0.5 mg/min for the remaining 18 hours).
- Lidocaine may be considered as an alternative to amiodarone for VF/pulseless VT that is unresponsive to CPR, defibrillation, and vasopressor therapy [Class IIb, level-of-evidence (LOE) C-LD]. Evidence does not support the routine use of a β -blocker after cardiac arrest; however, oral or IV β -blocker may be initiated or continued early after hospitalization from cardiac arrest due to VF/pulseless VT (Class IIb, LOE C-LD).
- Atropine should not be used during PEA or asystole as it is unlikely to have a therapeutic benefit.
- Other drugs not routinely used and should be considered.

Only in specific situations are:

- Magnesium sulfate (1–2 g) for hypomagnesemia and torsades de pointes associated with a long QT interval (25–50 mg/kg IV/IO over 10–20 minutes in children).
- Sodium bicarbonate (initial dose is 1 mEq/kg) repeat half this dose every 10 minutes thereafter. Should be used only if there is hyperkalemia (Class I), bicarbonate responsive acidosis or TCA overdose (Class IIa).

After prolonged resuscitation (IIb). It is harmful in hypercarbic acidosis (Class III).

- Calcium gluconate/chloride should not be routinely used. It is recommended only in the presence hyperkalemia, hypocalcemia or calcium channel blocker overdose.

Step 7

Advanced Airway

- Weigh the need for minimally interrupted compressions against the need for insertion of an advanced airway, i.e., ETT or supraglottic airway (laryngeal mask airway, esophageal tracheal tube or laryngeal tube).
- Continue bag mask ventilation if advanced airway is not placed.
- Confirm placement of advanced airway by clinical method (chest expansion and breath sound) and in addition use capnography to confirm and monitor correct placement. Record depth of insertion of ETT and secure it. Once the advanced airway is in place deliver one breath every 6 seconds (10 breaths/min) and continues chest compressions at a rate of 100–120/min independent of each other.

Step 8

Treat Reversible Causes

During each 2-minute period of CPR, review the most frequent causes—5H's and 5T's to identify factors that may have caused the arrest or may be complicating the resuscitation (**Table 1**).

TABLE 1: Reversible causes of cardiac arrest.

5H's	5T's
Hypovolemia	Tension pneumothorax
Hypoxia	Tamponade, cardiac
Hydrogen ion (acidosis)	Toxins
Hypo-/hyperkalemia	Thrombosis, pulmonary
Hypothermia	Thrombosis, coronary

Step 9

Monitor CPR Quality throughout Resuscitation

- Give emphasis on delivering high-quality CPR. This means giving compressions of adequate rate and depth, allowing complete chest recoil between compressions, minimizing interruptions in compressions, avoiding excessive ventilation and rotating the compressor every 2 minutes.
- Use quantitative waveform capnography to monitor end-tidal CO₂ (EtCO₂) (expressed as a partial pressure of EtCO₂ in mm Hg—P_{ET}CO₂) in intubated patients. If P_{ET}CO₂ < 10 mm Hg, attempt to improve CPR quality.

- If intra-arterial pressure monitoring is present then try to maintain the diastolic pressure > 20 mm Hg during chest compression.

The AHA defines chest compression fraction as a measurement of the proportion of time that compressions are performed during a cardiac arrest. More is the chest compression fraction, lesser are the interruptions in chest compressions. In adult cardiac arrest with an unprotected airway, chest compression fraction of at least 60% should be targeted.

It is reasonable to consider trying to optimize chest compression parameters if central venous oxygen saturation ($ScvO_2$) is $< 30\%$.

Routine measurement of arterial blood gases during CPR has uncertain value.

Step 10

Return of spontaneous circulation can be confirmed by return of pulse or blood pressure (BP) or abrupt sustained increase in $P_{ET}CO_2$ (typically ≥ 40 mm Hg) or spontaneous arterial pressure waves with intra-arterial monitoring.

Step 11

Postcardiac Arrest Care after ROSC

- **Goal:** Optimize cardiopulmonary function and vital organ perfusion.
- Transfer patient to an appropriate hospital or intensive care unit (ICU) with facility to deliver postcardiac arrest care.
- Optimize ventilation to minimize lung injury. Do chest X-ray to confirm secure airway and diagnose pneumonitis, pneumonia, pulmonary edema. Use lung-protective ventilation if there is pulmonary dysfunction; adjust settings using blood gas values. Avoiding excessive ventilation and hyperoxia. Once ROSC is achieved, the fraction of inspired oxygen (FiO_2) should be adjusted to the minimum concentration needed to achieve arterial oxyhemoglobin saturation $\geq 94\%$, with the goal of avoiding hyperoxia while ensuring adequate oxygen delivery.
- Treat hypotension [systolic BP (SBP) < 90] with fluid and vasopressor and treat the other reversible causes.
- Consider induced hypothermia. Adult patients with persistent coma after ROSC should be cooled to $32\text{--}34^\circ\text{C}$ for 12–24 hours provided SBP > 90 mm Hg or mean arterial pressure (MAP) > 70 mm Hg with or without vasopressors. Patients comatose before cardiac arrest or another reason to be comatose (e.g., drug overdose, status epilepticus) should be excluded. Cooling can be done using cold IV fluid bolus 30 mL/kg or using special endovascular catheters with feedback control to give cold fluids. This can be combined with surface cooling

using ice packs or mattresses. Sedation/muscle relaxants may be used to control shivering, agitation, or ventilator dyssynchrony as needed. After 24 hours, start slow rewarming at $0.25^\circ\text{C}/\text{h}$. Prevent hyperpyrexia $> 37.7^\circ\text{C}$. A recent high-quality study compared temperature management at 36°C and at 33°C postcardiac arrest and found outcomes to be similar for both. Hence, maintaining normothermia in these patients may be as effective and is a cheaper and less labor intensive strategy.

- Glucose control—moderate glycemic control (140–180 mg/dL). Use anticonvulsants if seizures present [use electroencephalogram (EEG) monitoring if available].
- Identify and treat acute coronary syndrome (ACS). Patients with suspected ACS should be sent to a facility with coronary angiography and interventional reperfusion facility [primary percutaneous coronary intervention (PCI)].
- Reduce the risk of multiorgan injury and support organ function, if required. **Flowchart 1** shows the adult cardiac arrest BLS algorithm for healthcare providers.

Q. What are the current guidelines for prognostication of a victim after cardiac arrest?

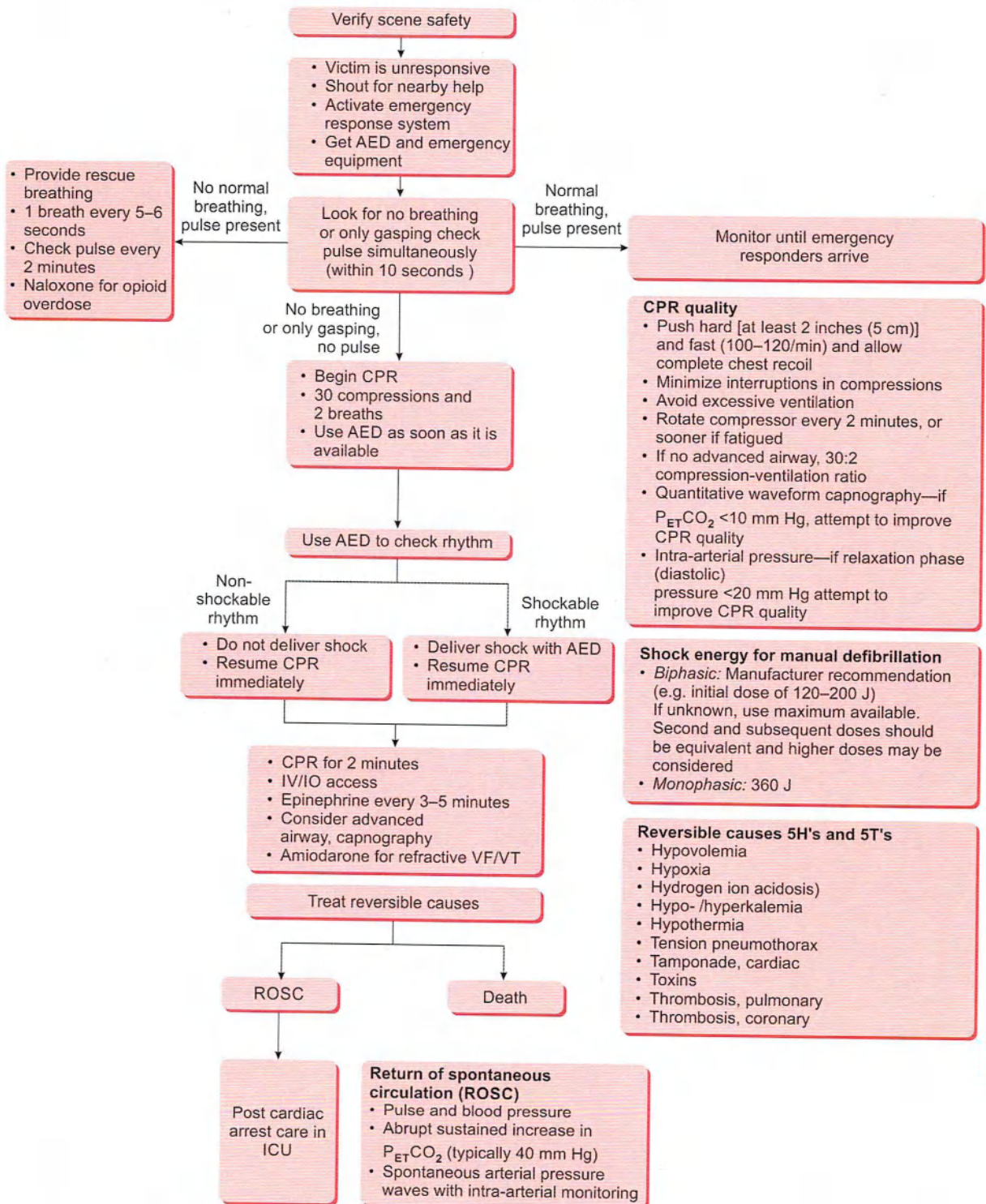
Ans. The earliest time to prognosticate a poor neurologic outcome using clinical examination is 72 hours after cardiac arrest in patients not treated with targeted temperature management (TTM). This may be done even later, if the residual effect of sedation or paralysis is suspected to confound the clinical examination. In patients treated with TTM, where sedation or paralysis could confound clinical examination, it is reasonable to wait until 72 hours after return to normothermia before predicting outcome.

No single physical finding or test can predict neurologic recovery after cardiac arrest with 100% certainty. Multiple modalities used together to predict outcome after the effects of hypothermia and medications have been allowed to resolve, are most likely to provide accurate prediction of outcome.

Clinical findings that are associated with poor neurologic outcome after cardiac arrest:

- Absence of pupillary reflex at 72 hours or more after cardiac arrest
- Presence of status myoclonus during the first 72 hours after cardiac arrest
- Absence of the N20 somatosensory evoked potential cortical wave 24–72 hours after cardiac arrest or after rewarming
- Presence of a marked reduction of the gray-white ratio on brain computed tomography (CT) obtained within 2 hours after cardiac arrest

Flowchart 1: Managing cardiac arrest.



(AED: automated external defibrillator; CPR: cardiopulmonary resuscitation; ICU: intensive care unit; IV: intravenous; IO: intraosseous; $P_{ET}CO_2$: partial pressure of end-tidal CO_2 ; VF: ventricular fibrillation; VT: ventricular tachycardia).

Source: Adapted from Kleinman ME, Brennan EE, Goldberger ZD, Swor RA, Terry M, Bobrow BJ, et al. Part 5: Adult basic life support and cardiopulmonary resuscitation quality: 2015 American Heart Association guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*. 2015;132(18 Suppl 2):S414–35.

- Extensive restriction of diffusion on brain magnetic resonance imaging (MRI) at 2–6 days after cardiac arrest
- Persistent absence of EEG reactivity to external stimuli at 72 hours after cardiac arrest
- Persistent burst suppression or intractable status epilepticus on EEG after rewarming.

Remember that shock, temperature, metabolic derangement, prior sedatives or neuromuscular blockers may affect results or interpretation of some tests.

Q. What is the adult chain of survival?

Ans. The AHA uses five links in a chain to illustrate the important time-sensitive actions in victims of SCA. Survival from cardiac arrest depends on a series of critical interventions; the term “chain of survival” summarizes the present understanding of the best approach to treat patients with SCA to optimize survival.

Links of in-hospital cardiac arrest (IHCA) chains of survival:

1. Surveillance and prevention
2. Recognition and activation of the emergency response system
3. Immediate high-quality CPR
4. Rapid defibrillation
5. Advanced life support and postarrest care.

Links of out of hospital cardiac arrest (OHCA) chains of survival:

1. Recognition and activation of the emergency response system
2. Immediate high-quality CPR
3. Rapid defibrillation
4. Basic and advanced emergency medical services (EMS)
5. Advanced life support and postarrest care.

Q. How does defibrillation work? What is an AED?

Ans. Defibrillation involves delivery of current through the chest and to the heart to depolarize myocardial cells simultaneously and eliminate VF. As soon as an AED/defibrillator is available, attach and defibrillate if indicated. Most adults with sudden witnessed nontraumatic cardiac arrest are found to be in VF; for these victims the time from collapse to defibrillation is the single greatest determinant of survival. Survival from VF arrest decreases by approximately 7–10% for each minute without defibrillation.

Automated external defibrillator is a computerized completely automatic defibrillator which uses voice and visual prompts to guide lay rescuers and healthcare providers to safely defibrillate. They can automatically analyze cardiac rhythm, charge to appropriate energy level and shock if advised. This is the single greatest advance in CPR which has significantly reduced in and out of hospital mortality after SCA. Due to the increasing evidence

for improved survival with early defibrillation and the introduction of AEDs, defibrillation which was earlier part of ACLS, has been now included as part of BLS. All AEDs are biphasic and are not recommended for use under the age of 1 year.

Q. What is the difference between defibrillation and cardioversion?

Ans. Cardioversion is the electrical maneuver that is used for organized rhythms, e.g., unstable tachycardia [supraventricular tachycardia (SVT), rapid atrial fibrillation (AF), etc.]. Here the timing of the shock with respect to cardiac cycle is important to avoid R on T phenomenon. The defibrillator/cardioverter has the ability to track the QRS, and to stick a visible marker on each one. This synchronization avoids shock delivery during the relative refractory period of the cardiac cycle when a shock could produce R on T phenomenon leading to VF. The same machine is used for both cardioversion and defibrillation; however, you have to select the “sync” button when you want to do cardioversion otherwise it will function as a defibrillator. Sedation will be required for cardioversion if the patient is awake as it is a painful and uncomfortable procedure. The recommended energy for cardioversion in atrial flutter is 50–100 J (biphasic), for AF is 120–200 J (biphasic) and for monomorphic VT it is 100 J (biphasic). In pediatric age group, energy recommendations for cardioversion are: 0.5–1 J/kg and can go up to 2 J/kg.

Defibrillation on the other hand is used only for the shockable cardiac arrest rhythms, i.e., VF and pulseless VT. Here there is no synchronization of the shock. Sedation is not required as all cardiac arrest victims are unconscious and the energy used is also much higher than during cardioversion.

Q. What is the difference between biphasic and monophasic defibrillation?

Ans. Modern defibrillators are classified according to two types of waveforms: (1) monophasic and (2) biphasic. Monophasic waveform defibrillators were introduced first, now biphasic waveforms are used in all AEDs and manual defibrillators sold today. Energy levels vary by type of device.

Monophasic waveforms deliver current of one polarity (i.e., direction of current flow). Monophasic waveforms can be further categorized by the rate at which the current pulse decreases to zero. The monophasic damped sinusoidal (MDS) waveform returns to zero gradually; whereas the monophasic truncated exponential (MTE) waveform current is abruptly returned to baseline (truncated) to zero current flow. Few monophasic waveform defibrillators are being manufactured but many are still in use.

Biphasic waveform delivers bidirectional current. Current research indicates, however, that when doses equivalent to or lower than monophasic doses (360 J) are used, biphasic waveform shocks are safe and effective for termination of VF. Research confirms that it is reasonable to use selected energies of 120–200 J with a biphasic truncated exponential (BTE) waveform. Outcome with the use of biphasic defibrillation is superior to monophasic defibrillation.

Biphasic waveform [BTE, rectilinear biphasic (RLB)] defibrillators are preferred to monophasic defibrillators for treatment of both atrial and ventricular arrhythmias due to greater chance of success in arrhythmia termination (Class IIa, LOE B-R).

Q. Why A-B-C has been changed to C-A-B in latest guidelines?

Ans. During the first minutes of VF SCA, rescue breaths are probably not as important as chest compressions because the oxygen level in the blood remains high for the first several minutes after cardiac arrest. In early cardiac arrest, myocardial and cerebral oxygen delivery is limited more by the diminished blood flow (cardiac output) than a lack of oxygen in the blood. Doing chest compression before ventilation saves 30 seconds. Both ventilations and compressions are important for victims of prolonged VF SCA, when oxygen in the blood is utilized.

During CPR, blood flow to the lungs is substantially reduced as the cardiac output is 25–33% of normal, so oxygen uptake from the lungs and CO₂ delivery to the lungs are also reduced. As a result, low minute ventilation (lower than normal tidal volume and respiratory rate) can maintain effective oxygenation and ventilation during CPR.

Excessive ventilation is unnecessary and is harmful because it increases intrathoracic pressure, decreases venous return to the heart, and diminishes cardiac output and survival. Avoid delivering breaths that are too large or too forceful. Such breaths are not needed and may cause gastric inflation and its resultant complications.

Q. Describe the special considerations during CPR in some special situations.

Ans.

• *Drowning:*

- Drowning is a preventable cause of death. The duration and severity of hypoxia sustained as a result of drowning is the single most important determinant of outcome.
- Rescuers should remove drowning victims from the water by the fastest means available and should begin resuscitation as quickly as possible.

- Provide CPR; particularly rescue breathing, as soon as the unresponsive submersion victim is removed from the water. Prompt initiation of rescue breathing increases the victim's chance of survival.
- Attempts to remove water from the breathing passages by any means other than suction (e.g., abdominal thrusts or the Heimlich maneuver) are unnecessary and potentially dangerous.
- Chest compressions are difficult to perform in water, may not be effective, and could potentially cause harm to both the rescuer and the victim.
- Only victims with obvious clinical signs of injury or alcohol intoxication or a history of diving, waterslide use, or trauma should be treated as a "potential spinal cord injury," with stabilization and possible immobilization of the cervical and thoracic spine.
- If vomiting occurs, turn the victim to the side.
- If defibrillation is required, dry the chest area well before applying the paddles.

• *Hypothermia:*

- Severe hypothermia [body temperature < 30°C (86°F)] is associated with marked depression of critical body functions, which may make the victim appear clinically dead during the initial assessment.
- In an unresponsive victim with hypothermia, assessments of breathing and pulse are particularly difficult because heart rate and breathing may be very slow, depending on the degree of hypothermia. Thus, lifesaving procedures should be initiated unless the victim is obviously dead (e.g., rigor mortis, decomposition, hemisection, decapitation). Do not wait to check the victim's temperature and do not wait until the victim is rewarmed to start CPR.
- Focus on interventions that prevent further loss of heat and begin to rewarm the victim immediately. To prevent further heat loss, remove wet clothes from the victim; insulate or shield the victim from wind, heat, or cold; and if possible ventilate the victim with warm, humidified oxygen.
- Victim should be transported to a center where aggressive rewarming during resuscitation is possible, as soon as possible.
- Passive rewarming is generally adequate for patients with mild hypothermia (temperature 34°C). For patients with moderate (30–34°C) hypothermia with a perfusing rhythm, external warming techniques are appropriate (forced air warming techniques). Passive rewarming alone will be inadequate for these patients. For patients with severe hypothermia (30°C) with a perfusing rhythm, core rewarming

is often used (warmed IV or IO fluids and warm humidified oxygen).

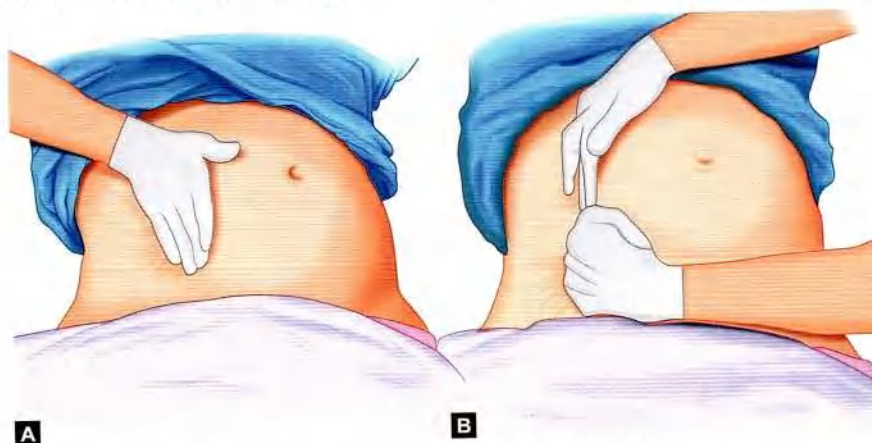
- If VF is detected, emergency personnel should deliver shocks using the same protocols used for the normothermic cardiac arrest victim.
- After ROSC, patients should continue to be warmed to a goal temperature of approximately 32–34°C for whom induced hypothermia is appropriate or normothermia should be targeted.
- **Pregnancy:**
 - Most common causes of maternal cardiac arrest are hemorrhage, cardiovascular diseases, amniotic fluid embolism, sepsis, aspiration pneumonitis, pulmonary embolism, and eclampsia.
 - Chest compressions should be given slightly higher than normal to adjust for the elevated diaphragm.
 - Priority for the pregnant victim is to provide high-quality CPR and remove aortocaval compression. The gravid uterus can compress the inferior vena cava, impeding venous return, thereby reducing stroke volume and cardiac output. If the fundus is at or above the level of the umbilicus, manual left uterine displacement should be given. This is done manually by one-hand or two-hand technique (**Figs. 1A and B**) or by using a wedge to give a 30° angle.
 - Anticipate difficult airway (due to modified anatomy and the tilt). Increased risk of aspiration can develop hypoxemia rapidly. They have decreased functional residual capacity and increased oxygen demand.
 - Defibrillation to be used as in nonpregnant adults.
 - IV should be secured above the level of diaphragm.
 - To simultaneously search and treat for the possible causes of cardiac arrest.
 - *Emergency cesarean delivery in cardiac arrest:* If there is no ROSC within 4 minutes; prepare for emergency cesarean section (hysterotomy). In an

obvious nonsurvivable injury, move straight to an emergency cesarean section. The best survival rate for the infant occurs when the infant is delivered no > 5 minutes after the mother's heart stops beating.

Q. How will you recognize a foreign body airway obstruction (FBAO) in an adult and how will you proceed with resuscitating such a victim?

Ans.

- Any FBAO should be recognized and relieved quickly.
- If mild obstruction is present and the victim is coughing forcefully, do not interfere with the patient's spontaneous coughing and breathing efforts. Attempt to relieve the obstruction only if signs of severe obstruction develop: the cough becomes silent, respiratory difficulty increases and is accompanied by stridor or the victim becomes unresponsive. Activate the EMS system quickly if the patient is having difficulty breathing.
- For responsive adults and children > 1 year of age with severe FBAO, case reports show the feasibility and effectiveness of back blows or slaps, abdominal thrusts and chest thrusts. The abdominal thrust is applied in rapid sequence until the obstruction is relieved. If abdominal thrusts are not effective, the rescuer may consider chest thrusts.
- Abdominal thrusts are not recommended for infants < 1 year of age because thrusts may cause injuries.
- Chest thrusts should be used for obese patients if the rescuer is unable to encircle the victim's abdomen and if the choking victim is in the late stages of pregnancy.
- If the adult victim with FBAO becomes unresponsive, the rescuer should carefully support the patient to the ground, immediately activate EMS and then begin CPR. Each time the airway is opened during CPR, the rescuer should look for an object in the victim's mouth and remove it. Simply looking into the mouth should not increase the time it takes to attempt the ventilations and proceed to the 30 chest compressions.



Figs. 1A and B: One-hand and two-hand technique.

- A finger sweep should be used only when the provider can see solid material obstructing the airway of an unresponsive patient as blind sweep can push the foreign body further in.

Q. What are the current recommendations regarding the route of drug administration during CPR?

Ans. Peripheral venous access is the procedure of choice during CPR; cannulate the large easily accessible peripheral veins like the cephalic or external jugular vein. During cardiac arrest, follow all administered drugs with a bolus of 20 mL IV saline while using a peripheral line and a limb raise. Use a central line if already in place, do not attempt central venous cannulation if peripheral access is available as this requires asepsis, skills and takes more time than taking a peripheral line.

If IV access cannot be established, IO delivery of resuscitation drugs will achieve adequate plasma concentrations (You will need special IO needles for this). Resuscitation drugs can also be given via the tracheal tube, but the plasma concentrations achieved are variable and substantially lower than those achieved when the same drug is given by the IV or IO route. If IV access is delayed or cannot be achieved, IO access should be considered.

Q. What are the current recommendations for the use of vasopressors during cardiac arrest?

Ans. Vasopressin has been removed from the ACLS cardiac arrest algorithm to simplify the approach as epinephrine is recognized to have equivalent effect.

Epinephrine is indicated for all cardiac arrest rhythms, i.e., VF, pulseless VT, asystole and PEA. IV/IO dose is 1 mg administered every 3–5 minutes followed by 20 mL saline flush. In large randomized clinical trials, doses of epinephrine > 1 mg were not beneficial and did not improve survival. There is no upper limit on how much adrenaline can be given.

Q. Which are the investigations you will perform in a cardiac arrest victim during resuscitation?

Ans. Immediate investigations:

- Arterial blood gas (to look for acidosis)
- Blood glucose
- Serum electrolytes (to look for hyperkalemia).

Q. What is impedance threshold device (ITD)?

Ans. The ITD is a valve that limits air entry into the lungs during chest recoil between chest compressions. It is designed to reduce intrathoracic pressure and enhance venous return to the heart. The addition of the ITD improved the hemodynamics during standard CPR in five laboratory studies and one clinical study. It is not in regular use. Only centers with expertise may use it as an adjunct.

Q. How does chest compression help during CPR? What alternate techniques and devices other than standard chest compression that have been described to assist circulation and what is their current status?

Ans. Chest compressions create blood flow by increasing intrathoracic pressure or by directly compressing the heart. When rescue breathing is provided using oxygen and blood is circulated to the lungs by chest compression the victim will receive oxygenation to the brain and the other vital organs. "Effective" chest compressions are essential for providing blood flow during CPR (Class I). To give "effective" chest compressions, "push hard and push fast".

Open-Chest CPR

Open-chest CPR is not recommended for routine use. It should be considered only for special situations, such as cardiac arrest in cardi thoracic surgery, patients on table or in the early postoperative phase after or when the chest or abdomen is already open as during surgery or in case of penetrating chest trauma.

Prone CPR

When the patient cannot be placed in the supine position, rescuers may consider providing CPR with the patient in the prone position, particularly in patients with an advanced airway in place, like in the operating room. The chest compressions are given with the hands of the rescuer in the midline between the two scapulae (i.e., at T7–10 position) with rest of the technique same as for supine CPR. Several cases series have documented survival to discharge when this technique was used.

Compression-Only CPR

Recent studies suggest improved outcome toward higher compression rates and more time spent for compression with less emphasis on ventilation.

The outcome of chest compressions without ventilations is significantly better than the outcome of no CPR for adult cardiac arrest. Laypersons should be encouraged to do compression-only CPR if they are unable or unwilling to provide rescue breaths (Class IIa), although the best method of CPR is compressions coordinated with ventilations.

Other Techniques and Devices

- Interposed abdominal compression (IAC) CPR.
- Active compression-decompression (ACD) CPR.
- *Load-distributing band (LDB) CPR*: The LDB is a circumferential chest compression device composed of a pneumatically actuated constricting band and backboard.
- Mechanical (Piston) CPR.
- *Lund University Cardiopulmonary Assist System (LUCAS) CPR*: The LUCAS is a gas-driven sternal

compression device that incorporates a suction cup for active decompression.

- *Phased thoracic-abdominal compression-decompression (PTACD) CPR*: The PTACD-CPR combines the concepts of IAC-CPR and ACD-CPR.
- *Minimally invasive direct cardiac massage (MIDCM)*: The MIDCM involves insertion of a plunger-like device through a small incision in the chest wall to enable direct compression of the heart.

Many of these devices and techniques have shown promising results both in laboratory and human studies. However, there is insufficient data to support its use for routine resuscitation.

Q. Describe the rate and technique of chest compression for infants and children.

Ans. If there is no pulse or pulse is <60 bpm with signs of poor perfusion (with no breathing or gasping), begin chest compressions. While giving chest compression “push hard and push fast” at a rate of 100–120 times per minute to depress the chest to $1/3$ rd AP diameter or 2 inches (5 cm) in children and 1.5 inches (4 cm) in infants. Release completely to allow full recoil of the chest. Minimize interruptions to chest compression. Compression ventilation ratio of 30:2 (one rescuer) and 15:2 (two rescuers) to be maintained.

In Infants

- Two-finger chest compression technique (one rescuer)—compress the sternum with two fingers placed below the intermammary line (**Fig. 2**).

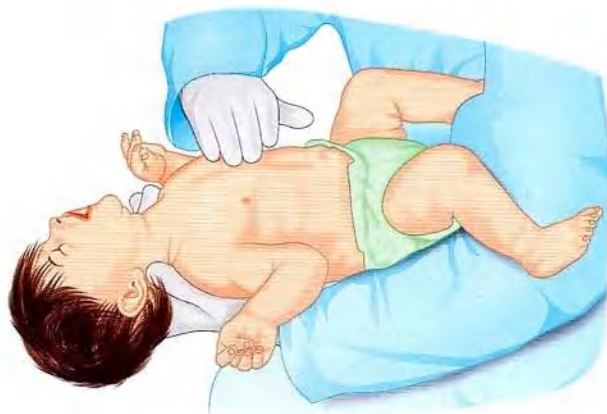


Fig. 2: Two-finger chest compression technique in infant (one rescuer).

- Two-thumb-encircling technique (two rescuers)—encircle the infant's chest with both hands, spread your fingers and place your thumbs together over the lower-half of the sternum and forcefully compress the sternum with your thumb (**Fig. 3**).

The two-thumb encircling technique is recommended when two rescuers are present. It is also preferred because it produces better coronary perfusion and may generate higher systolic and diastolic pressures.



Fig. 3: Two-thumb-encircling hands chest compression in infant (two rescuers).

In Child

Compress the lower end of the sternum with the heel of one hand or two hands but should not press on the xiphoid or the ribs.

Q. Which are the common causes of cardiac arrest under anesthesia?

Ans. Cardiac arrest can occur during induction, maintenance, intraoperatively or in postoperative period. It is more common during induction and reversal.

Cardiac arrest can occur anytime during the course of the anesthetic. The causes are related to:

- *Airway*:
 - “Cannot ventilate cannot intubate” is a dreaded complication. Rescue airway devices should be immediately used.
 - Many airway-related problems may occur during the course of anesthetic like blocked tube, disconnections, displacement of tube. Continuous EtCO₂ is helpful in recognizing these problems early.
 - Premature extubation if unrecognized.
 - *Foreign body*: Any unrecognized foreign body in the oropharynx, e.g., throat pack, dislodged tooth.
- *Breathing*:
 - Laryngospasm at induction or postextubation.
 - Intraoperative/postoperative bronchospasm when severe can cause hypoxia and bradycardia leading to cardiac arrest.
 - *Tension pneumothorax*: Patients with preexisting bullae or emphysema are at risk.

- **Aspiration:** It can occur at induction or postextubation.
- **Residual anesthetic effect:** Residual narcotic/inhalation effect or residual paralysis can lead to hypoventilation and hypoxemia.
- **Pulmonary edema:** Due to fluid overload/aspiration.
- **Circulation:**
 - **Embolism:** Pulmonary thromboembolism, e.g., air (laparoscopic, neurosurgery), fat (rimming of long bones), amniotic fluid (at placental separation), tumor (cancer surgeries). Continuous EtCO₂ and BP monitoring is helpful in recognizing the problem.
 - Perioperative myocardial infarction.
 - Massive uncontrollable perioperative blood loss.
 - **Vasovagal:** Generally occurs in young individuals with high vagal tone.
 - Patients for emergency surgery with severe cardiac depression in systemic inflammatory response syndrome (SIRS)/sepsis.
- **Drug induced:**
 - Succinylcholine-induced hyperkalemia.
 - **Anaphylaxis:** To drugs/fluids.
 - Accidental intravascular or intrathecal injection of local anesthetic.

Q. What is the mechanism of cardiac arrest under spinal anesthesia?

Ans. Decrease in preload due to vasodilatation causes reflex severe bradycardia. In patient with high vagal tone, this severe bradycardia leads to cardiac arrest.

Q. What is the treatment of postspinal bradycardia?

Ans. Atropine 0.4–0.6 mg is first drug of choice. Glycopyrrolate is ineffective, hence not recommended.

If bradycardia is not reverted with atropine; ephedrine (25–50 mg), or epinephrine (0.2–0.3 mg) may be considered.

If bradycardia leads to cardiac arrest, follow CPR flow chart.

Q. What are the causes of cardiac arrest in pediatric patient?

Ans.

- Airway and breathing problems are the main cause of cardiac arrest in pediatric patients.
 - In infants, the leading causes of death are congenital malformations, complications of prematurity, and sudden infant death syndrome (SIDS).
 - In children over 1 year of age, injury/accident is the leading cause of death.
 - Cardiac causes predominantly hypovolemia from blood loss is one of the causes of cardiac arrest in children especially in patients of spinal fusion or craniotomy.
- Hyperkalemia following blood transfusion and severe laryngospasm can sometimes lead to cardiac arrest in children.

Q. Describe the pediatric BLS sequence.

Ans. Flowchart 2 shows the pediatric BLS sequence.

Q. Enumerate the key differences in between adult, pediatric and infant BLS.

Ans. Table 2 shows the differences between adult, pediatric and infant BLS.

TABLE 2: Differences between adult, pediatric and infant basic life support (BLS).

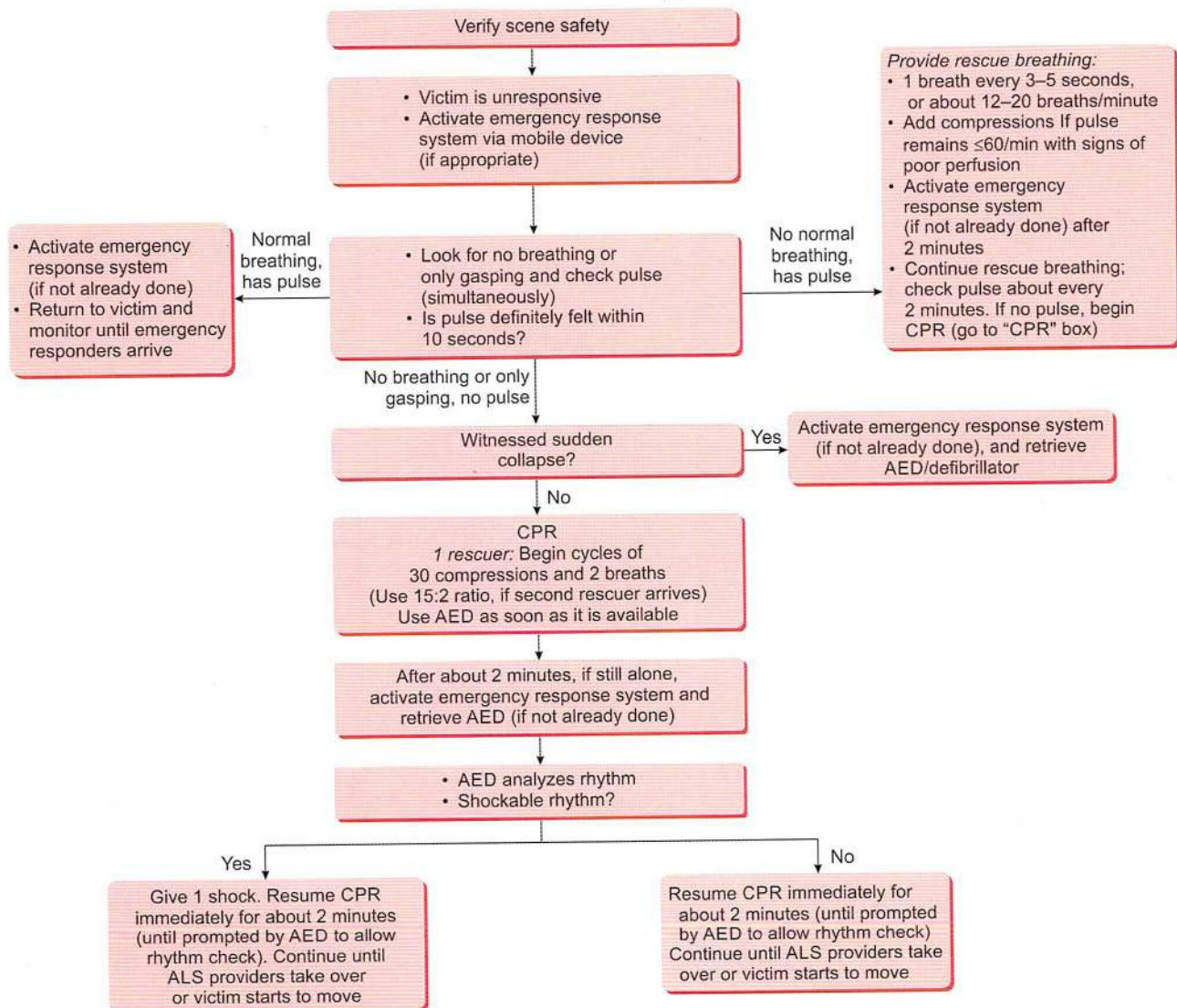
Recommendations			
Component	Adult	Child (1 year to puberty)	Infant (<1 year)
Recognition	Unresponsive		
	No breathing or no normal breathing (i.e., only gasping)	No breathing or only gasping	
	No pulse palpated within 10 seconds		
CPR sequence	C-A-B		
Compression technique	Heel of both hand	Heel of one hand	Two-finger chest compression technique/ two-thumb-encircling technique
Compression rate	100–120/min		
Compression depth	At least 2 inches (5 cm)	• At least 1/3rd AP diameter • About 2 inches (5 cm)	• At least 1/3rd AP diameter • About 1–1.5 inches (4 cm)
Chest wall recoil	Allow complete recoil between compressions rotate compressors every 2 minutes		
Compression interruption	Minimize interruptions in chest compressions limit interruptions to <10 seconds		
Airway	Head tilt-chin lift (suspected trauma: jaw thrust)	Head tilt-chin lift (both in injured and noninjured victims)	
Compression-to-ventilation ratio (until advanced airway placed)	30:2 (one or more rescuers)	30:2 (single rescuer) 15:2 (two rescuers)	

Contd...

Contd...

Recommendations		
Ventilations with advanced airway	One breath every 6 seconds (10 breaths/min)	One breath every 6–8 seconds (8–10 breaths/min)
	Asynchronous with chest compressions About 1 second per breath Visible chest rise	
Defibrillation	• Attach and use AED as soon as available • Minimize interruptions in chest compressions before and after shock • Resume CPR beginning with compressions immediately after each shock	
	• Biphasic: 120–200 J • Monophasic: 360 J	• 2–4 J/kg first and 4 J/kg for subsequent shocks, higher energy may be considered but not to exceed 10 J/kg

Flowchart 2: Pediatric basic life support sequence.



(ALS: advanced life support; AED: automated external defibrillator; CPR: cardiopulmonary resuscitation).

Source: Adapted from Kleinman ME, Brennan EE, Goldberger ZD, Swor RA, Terry M, Bobrow BJ, et al. Part 5: Adult basic life support and cardiopulmonary resuscitation quality: 2015 American Heart Association guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*. 2015;132(18 Suppl 2):S414-35.

SUGGESTED READING

1. Bhanji F, Donoghue AJ, Wolff MS, Flores GE, Halamek LP, Berman JM, et al. Part 14: Education: 2015 American Heart Association Guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*. 2015;132(18 Suppl 2):S561-73.
2. Biboulet P, Aubas P, Dubourdieu J, Rubenovitch J, Capdevila X, d'Athis F. Fatal and non-fatal cardiac arrests related to anesthesia. *Can J Anaesth*. 2001;48:326-32.
3. Hazinski MF. Highlights of the 2010 AHA Guidelines for CPR and ECC—American Heart Association; 2010.
4. Kawashima Y, Takahashi S, Suzuki M, Morita K, Irita K, Iwao Y, et al. Anesthesia-related mortality and morbidity over a 5-year period in 2,363,038 patients in Japan. *Acta Anaesthesiol Scand*. 2003;47:809-17.
5. Kilgannon JH, Jones AE, Shapiro NI, Angelos MG, Milcarek B, Hunter K, et al. Association between arterial hyperoxia following resuscitation from cardiac arrest and in-hospital mortality. *JAMA*. 2010;303:2165-71.
6. Morray JP, Geiduschek JM, Ramamoorthy C, Haberkern CM, Hackel A, Caplan RA, et al. Anesthesia-related cardiac arrest in children: initial findings of the Pediatric Perioperative Cardiac Arrest (POCA) Registry. *Anesthesiology*. 2000;93:6-14.
7. Newland MC, Ellis SJ, Lydiatt CA, Peters KR, Tinker JH, Romberger DJ, et al. Anesthetic-related cardiac arrest and its mortality: a report covering 72,959 anesthetics over 10 years from a US Teaching Hospital. *Anesthesiology*. 2002;97:108-15.
8. Pollard JB. Cardiac arrest during spinal anesthesia: common mechanisms and strategies for prevention. *Anesth Analg*. 2001;92:252-6.
9. Sprung J, Warner ME, Contreras MG, Schroeder DR, Beighley CM, Wilson GA, et al. Predictors of survival following cardiac arrest in patients undergoing noncardiac surgery: a study of 518,294 patients at a tertiary referral center. *Anesthesiology*. 2003;99:259-69.

Airways, Connectors, Laryngoscopes, and Non-rebreathing Valves

Nayana Amin, Sudivya Sharma, Sukhada Savarkar

AIRWAYS

Q. Define an airway.

Ans. An airway is a device which when inserted into either the oropharynx or the nasopharynx helps to maintain the patency of the air passage for unobstructed breathing.

There are various maneuvers such as chin lift, jaw thrust, and tracheal intubation which help to maintain a patent airway. However, unlike these maneuvers, insertion of an airway does not affect the stability of the cervical spine.

Q. How are airways classified?

Ans. Airways are classified as:

- Oropharyngeal airways
- Nasopharyngeal airways
- *Modified airways:*
 - Laryngeal mask airway
 - Cuffed oropharyngeal airway.

Also, artificial airways may be simple supraglottic devices (oropharyngeal and nasopharyngeal airways), augmented supraglottic devices (laryngeal mask airway

and airway management devices), and infraglottic devices (endotracheal tubes and tracheostomy tubes).

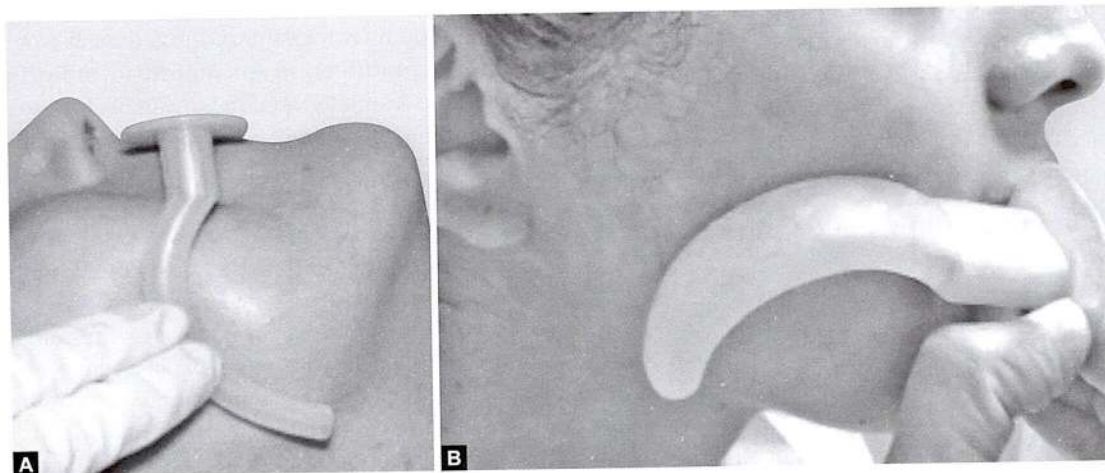
Q. What are the uses of airways?

Ans.

- Prevent obstruction of the upper air passage by lifting the tongue and epiglottis away from the posterior pharyngeal wall
- Prevent biting of and occlusion of the orotracheal tube
- Protect the tongue during biting and seizure activity
- Facilitate oropharyngeal suctioning
- Provide a better mask fit for ventilation
- Help insertion of tubular devices (suction catheters and Ryles tube) into pharynx or esophagus.

Q. Give a brief description of an oropharyngeal airway.

Ans. It is a suitably curved apparatus made of metal, plastic, or hard rubber. There is a flange at the proximal end which can be used to fix the airway. The flange also prevents the airway from moving deeper into the mouth. The bite portion is straight and firm so that the patient cannot close the air



Figs. 1A and B: Oropharyngeal airway size determination.

channel by biting hard. When an appropriate sized airway is in place, the bite portion lies between the lips and teeth and the flange lies outside the lips. The pharyngeal end is the curved portion of the airway that extends upward and backward to correspond to the shape of the tongue and palate. It separates the tongue from posterior pharyngeal wall and it also pulls the epiglottis slightly forward by exerting pressure along the base of the tongue. When inserted, the distal end should lie just above the epiglottis so as to not irritate the laryngeal inlet. The cross-section of an oropharyngeal airway is either oval or circular in shape. These airways are available in various lengths and diameters so that they can be used in neonates as well as in adults (Figs. 1A and B).

Q. How do you estimate the appropriate size of an oropharyngeal airway? Describe the insertion technique.

Ans. An estimate of the appropriate-sized airway is obtained by selecting an airway with the length corresponding to the vertical distance between the patient's incisor and the angle of the jaw. If the airway is too small, it may cause kinking of the tongue and obstruct the movement of gas. If an airway is too large, it may cause trauma to the larynx or it may displace the epiglottis and cause airway obstruction.

Insertion Technique

Open the patient's mouth and insert a well-lubricated airway into the oral cavity with its curvature facing the upper lips. Once it is halfway down, it is rotated to its normal position and advanced further until the distal end lies in the oropharynx. This rotation technique minimizes the chance of pushing the tongue backward and downward. Jaw thrust can be given to overcome any resistance during insertion. Another method of insertion is to depress the tongue with a tongue depressor and insert the airway with its concavity facing the tongue. The flange is pushed to slide the airway behind the tongue. If the patient's jaw is not supported, it may cause the airway to be pushed out. However, if the airway repeatedly comes out, it should be replaced with a smaller sized airway. Correct placement is shown by improvement in airway patency and by seating of the flattened reinforced section between the patient's teeth or alveolar margins, if the patient is edentulous.

Q. Give a brief description of the various types of oropharyngeal airways.

Ans.

Guedel Airway (Sizes—0–6)

Description: It is made up of rubber. It is a hollow oropharyngeal airway with a large flange and a reinforced bite portion. It has a gentle curve that follows the contour of the tongue. It has a tubular channel for air exchange and suction. It is most frequently used.

Sterilization: Cleaning with soap and water. However, repeated sterilization may cause softening.

Problems: Latex allergy. Softening may allow the lumen to be occluded and not prevent the tube from biting.

Waters Airway (Sizes—00, 0, 1 to 7)

Description: It is named after Ralph Waters. It is a metallic oropharyngeal airway with two holes at the pharyngeal end. It is hollow and has a right or left nipple on the flange for attachment of oxygen line and suction.

Sterilization: Cleaning with soap and water.

Advantages: It is cheap, reusable, and easy to store.

Connell Airways (Sizes—00, 0, 1 to 7)

Description: It is a metallic hollow oropharyngeal airway. It is similar to Waters airway but without the insufflation nipple.

Berman Airway (Sizes—Infant, Small Child, Child, Medium Adult, Large Adult)

Description: It has a flange at the buccal end. It has no enclosed air channel. The sides are cut-open and there is support through the center (H-shaped in cross-section) through which a tracheal tube can be passed. The sides allow passage of suction catheters and provide air channels. The center may have openings in it to permit suction if the airway becomes lodged sideways. It can be used as an aid for oral flexible bronchoscopy.

Advantage: It is easier to clean and is less likely to become obstructed with foreign body or mucus.

Q. Which airways can be used for resuscitation if you do not have manual resuscitator? Can you describe any one of them?

Ans.

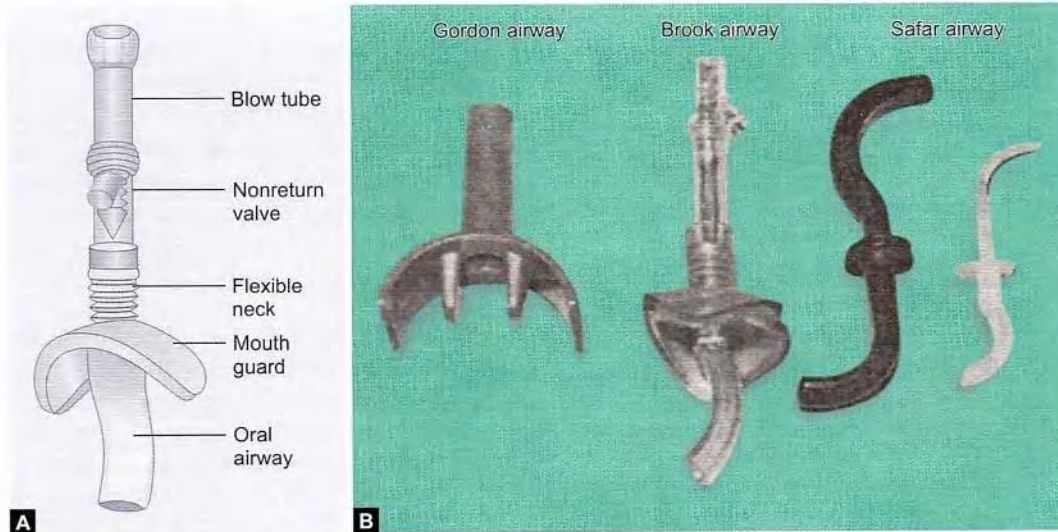
Safar Airway (Sizes—Adult and Pediatric)

Description: In 1958, Safar and McMahon described an S-shaped oropharyngeal airway which consisted of two Guedel-type airways soldered together. It is a new and aesthetic modification for mouth-to-mouth artificial respiration. A smaller version is available for use in infants and children. It is made of nontraumatic soft rubber.

Use: Mainly for artificial resuscitation.

Brook Airway

Brook airway was devised by Morris Brook in 1957. Like Safar, he started with a Guedel airway to which he attached a mouth guard flap which fit snugly over the patient's lips. A flexible rubber neck with a straight plastic tube was attached to the mouth guard. The straight tube had a built-in spring one-way valve which allowed air to be blown into the tube, but at the same time, the expired air from the victim would escape via the valve through a side exit. The advantage of



Figs. 2A and B: Various types of airway. (A) Brook airways; (B) Resuscitation airways.

Brook's airway was the flexible neck which allowed the tube to be angled if necessary. A suction channel with an attached suction-bulb has been added in the later models.

Various types of airways are shown in **Figures 2A and B**.

Q. Which airways are used as an aid for fiberoptic intubation?

Ans.

Ovassapian Fiberoptic Intubating Airway

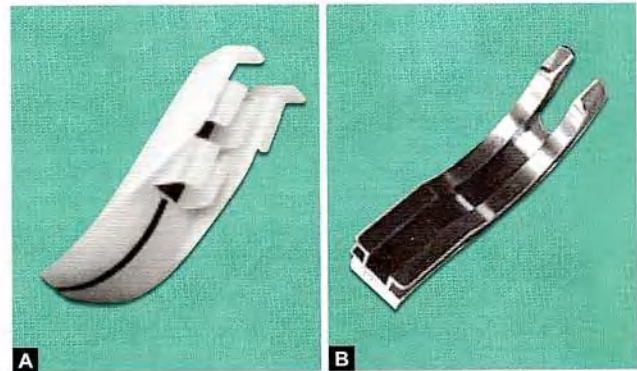
Ovassapian fiberoptic intubating airway is designed for use during fiberoptic intubation.

Description: The proximal end is flat and narrow and functions as a bite block. The distal end gradually widens. It has two vertical side walls at the buccal end. Between the side walls, there are two pairs of curved guide walls which allow the passage of a tracheal tube up to 9 mm ID. The guide walls are flexible so that the airway can be removed from around the tracheal tube after the intubation has been completed. The proximal end is tubular so that it can function as a bite block. Since, there is no posterior wall in the distal half of the airway, it is easy to maneuver the fiberoptic scope in the oropharynx. Also, during fiberoptic intubation, it is not necessary to remove the tracheal tube connector when using this airway (**Fig. 3A**).

Patil-Syracuse Oral Airway or Patil Endoscopic Airway

Patil-Syracuse oral airway or Patil endoscopic airway was designed as an aid to fiberoptic intubation.

Description: It has suction channels at the sides. On the lingual surface, there is a groove at the center which allows the passage of the fiberscope and guides it in the midline. The distal end has a slit which allows the fiberscope to be manipulated in the anteroposterior direction (**Fig. 3B**).



Figs. 3A and B: (A) Ovassapian airway; (B) Patil-Syracuse oral airway.

Berman Intubating Airway

Description: It is tubular along its entire length. It is open on one side so that it can be split and removed from around a tracheal tube.

Williams Airway Intubator

Description: It is designed for blind orotracheal intubation and also used as an aid in fiberoptic-guided intubation. The proximal half is cylindrical and distal half is open on the lingual side.

Esophageal Obturator Airway

Description: This airway has a face mask at the proximal end, which fits over the mouth and nose of the victim. There is a blowing device in the center of the facemask through which air or oxygen can be blown during artificial respiration. This blowing tube continues below as a curved and elongated hollow tube with a blind end. There is an esophageal balloon just before the blind end. The balloon needs to be inflated with 10–20 mL of air to obliterate the esophagus and prevent regurgitation and aspiration. There are multiple small holes

along the walls of the tube. The air that is blown through the tube passes through these holes to enter the patient's lungs (Fig. 4).

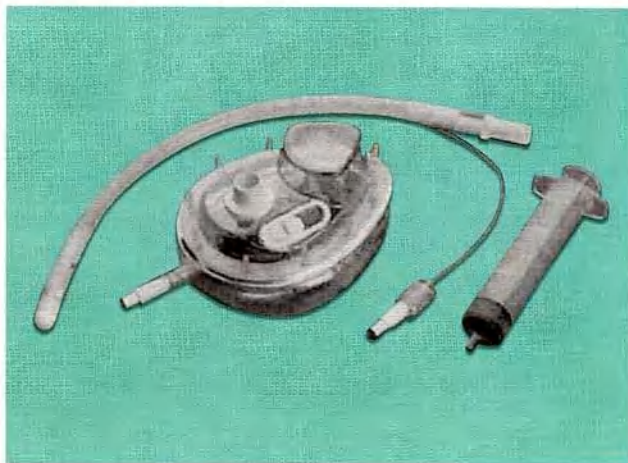
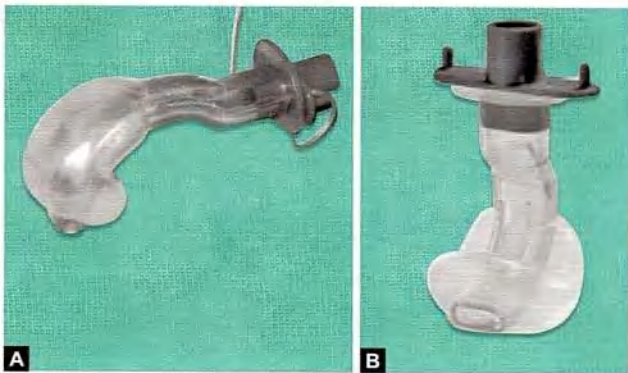


Fig. 4: Esophageal obturator airway.

Q. Which oropharyngeal airways can be used for intermittent positive pressure ventilation (IPPV)?

Ans. Cuffed oropharyngeal airway (Figs. 5A and B) and laryngeal mask airway can be used for IPPV.

Use: This is used for artificial resuscitation.



Figs. 5A and B: Cuffed oropharyngeal airway.

Q. What is cuffed oropharyngeal airway?

Ans. Cuffed oropharyngeal airway is Guedel airway with inflatable cuff designed to seal the airway. It has an integral bite block and 15 mm connector for connecting to the breathing circuit. The cuff is inflated with air to displace the base of tongue and forms a low pressure seal and provides an open airway.

Q. What are the advantages of a nasopharyngeal airway over an oropharyngeal airway?

Ans. Nasal airway is better tolerated in a semiawake patient than an oral airway and is less likely to be accidentally displaced or removed.

They offer an alternative to the oral airway when the patient has limited mouth opening, awkward or fragile dentition, trauma or pathology of oral cavity, or where oral airway is frequently displaced by marked overlapping bite.

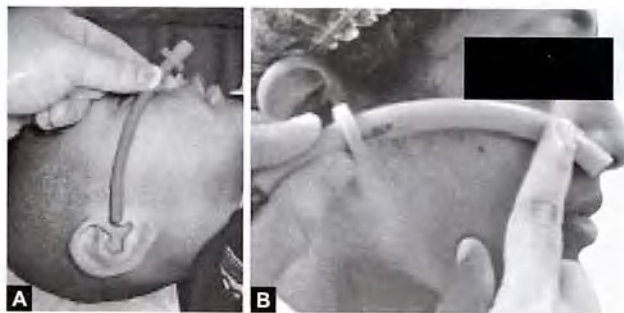
They have also been used to aid in pharyngeal surgery, to reduce trauma while passing fiberoptic bronchoscope, to apply continuous positive airway pressure (CPAP), to facilitate suctioning, and to help in airway management of a patient with Pierre Robin syndrome.

Q. Give a brief description of a nasopharyngeal airway.

Ans. Nasopharyngeal airways are made of soft plastic, polyurethane, or latex rubber. At its proximal end, there is either a fixed or adjustable flange which limits the insertion of an excessive length so that the device lies just above the epiglottis. The tube is curved to fit the curvature of the nasopharynx. A beveled distal end makes its passage through the nose less traumatic. Sometimes, there may be a hole cut into the wall opposite the bevel which ensures airway patency, even if the bevel becomes blocked with mucus during insertion. It is available in a range of lengths and internal diameters.

Q. How would you choose an appropriate size nasopharyngeal airway? Describe the insertion technique.

Ans. The length of the airway needed for the patient can be estimated as the distance from the tragus of the ear to the tip of the nose plus 1 inch or the distance from the tip of the nose to the meatus of the ear (Figs. 6A and B).



Figs. 6A and B: Measurement of nasal airway.

Insertion Technique

Before insertion, it should be lubricated along its entire length. Use of vasoconstrictors before the insertion of the airway may not always be beneficial. Check the patency of the right nostril. The airway is held in the hand on the same side as it is to be inserted. It is inserted through the nares, bevel end first. The bevel should face the septum to reduce trauma. It is passed vertically along the floor of the nose with slight twisting action. The curve of the airway should be directed toward the patient's feet. If resistance is encountered, the airway should be redirected. If excessive

resistance is encountered, the other nostril or smaller airway should be used. When properly placed, it extends from the nose to the pharynx with the pharyngeal end above the epiglottis and below the base of the tongue and with the flange just outside the nostrils.

Q. What are the various types of nasopharyngeal airways that you know of?

Ans. Bardex airway: It is made of rubber. It has a large flange at the nasal end and a bevel at the pharyngeal end.

Rusch airway: It is made of red rubber. It has a short bevel at the pharyngeal end and an adjustable flange at the nasal end.

Saklad airway: It is almost like Bardex airway, except that it does not have a circular flange in the proximal end, making it vulnerable to slip into the oropharynx.

Linder nasopharyngeal airway: It is a clear plastic airway with a large flange. The distal end is flat rather than beveled. The airway is supplied with an introducer which has a balloon. A Luer lock syringe attached to a one-way valve at the other end of the introducer can be used to inflate or deflate the balloon.

Binasal airway: This airway consists of two nasal airways joined together by a connection that has a 15 mm adaptor for attachment to the breathing system. The tube is positioned so that the distal tips are near the larynx just below the tip of the epiglottis. After the airway is inserted, the soft tissues usually seal the hypopharynx permitting assisted or controlled respiration.

Q. What are the precautions to be taken during the insertion of an airway?

Ans.

- Pharyngeal and laryngeal reflex should be depressed to prevent coughing and laryngospasm.
- There should be selection of correct size.
- The airways should be well lubricated before insertion.

Q. Which airway is used during electroconvulsive therapy?

Ans. During electroconvulsive therapy (ECT), the oral airway inserted serves the purpose of preventing tongue bite more than acting as an airway, as the shock is given after preoxygenation and patient is apneic during the seizure. A simple bite block or Oberto mouth prop can be used.

Q. What are the problems that can arise during the use of an airway?

Ans.

- Airway obstruction—too large an airway, foreign body may be lodged in the air channel.

- Airway can be compromised due to epistaxis resulting from trauma to the septal mucosa, nasal polyps, or adenoidal tissue during insertion of nasopharyngeal airway.
- Trauma to the tongue, nose, uvula, pharynx, and teeth.
- Long oropharyngeal or nasopharyngeal airway can cause laryngospasm.
- Insertion of oropharyngeal airway in light planes of anesthesia can result in gagging or retching.
- Latex allergy.

Q. How are the airways sterilized?

Ans. Airways are cleaned with soap and water.

CONNECTORS

Q. What is the difference between a connector and an adaptor?

Ans. A connector is a fitting intended to join together two or more components. An adaptor is a specialized connector that establishes functional continuity between otherwise disparate or incompatible components. An adaptor or a connector may be distinguished by:

- Shape (straight, right angle or elbow, T or Y)
- Components to which it is semipermanently attached
- Added feature (with nipple or pop-off)
- Size and type of fitting at either end (15 mm for male and 22 mm for female).

All anesthesia systems terminate at the patient end by connection to a mask or endotracheal tube adaptor. All face masks, both adult and pediatric, have a 22 mm for female and a 15 mm for male fitting.

Hence, connectors should have:

- 15 mm male fitting at the machine end
- 22 mm male/15 mm female fitting for connection to either tracheal tube adaptor or a mask at the patient end
- 15 mm female fitting for connection to a tracheal tube adaptor.

Often this component is a right-angle connector and may be known as elbow adaptor, elbow joint, elbow connector, face mask angle piece, mask adaptor, or mask elbow.

Q. What are their uses?

Ans.

- To extend the distance between the patient and the breathing system (head and neck surgery)
- To change the angle of connection between the tracheal tube and the breathing system
- To allow a more flexible and/or less kinkable connection between the tracheal tube and the breathing system.

Q. What are the principles to be kept in mind while selecting a connector?

Ans. While selecting a connector, several principles should be kept in mind. These are as follows:

- Connectors increase dead space (significant in infants).
- Addition of a connector increases the number of possible locations at which disconnections can take place.
- Connectors with sharp curves and rough side walls (Cobb's suction union and Rowbotham adaptors) can markedly increase the resistance.
- Straight connectors offer less resistance than corrugated connectors; hence, to minimize the resistance, endotracheal tube adaptors should preferably be straight or gently curved.

Q. Describe a tracheal tube connector.

Ans. It is a 15 mm male-to-female ISO tapered connection used to connect the endotracheal tube to the components of the breathing system. The male connector has a tapered cone which is pushed into the lumen of the endotracheal tube. While fitting the connector, the tube material has to be stretched as it is slightly bigger than the tube. This helps to produce a more secure connection.

Q. What is the difference between adult and pediatric connectors?

Ans. Prior to any standardization, the sizes of the connector depended on the manufacturer (Magill connectors, 13 mm; Worcester, 12 mm; Metal Cardiff, 12 mm; Knight, 9 mm; and Bennett, 14 mm). However, subsequently a 15 mm ISO connection was standardized for use in adults and large children. Recently, 8.5 mm connectors are available for endotracheal tube sizes 2–6 mm ID as the standard 15 mm ISO connector may not be suitable for use in infants and neonates. However, these (8.5 mm) connectors will not fit our standard 15 mm ISO connector.

Q. What are the ISO recommendations for tapered connections or adaptors?

Ans. Tapered connections or adaptors are used to join rigid tubes or other components in such a manner that the joint will not leak. This joint has a male-half and a female-half which is pushed together with a slight twist to form a gas tight fit. These connectors and adaptors should be easy to dismantle and reassemble.

Current ISO recommendations are as follows:

- 30 mm tapered connections for attachment of scavenging hose to breathing systems
- 22 mm tapers for connection within the breathing system
- 15 mm connection for attachment of breathing system to an endotracheal tube.

Q. How would you clean and sterilize the connectors?

Ans. Cleaning: After using connectors, it should be rinsed under a running tap, then placed in a solution of detergent and water and soaked.

Disinfection and sterilization: Rubber and plastic connectors may be sterilized with ethylene oxide or in a liquid such as glutaraldehyde. Metal connectors may be boiled, autoclaved, or pasteurized.

■ LARYNGOSCOPES**Q. How would you define a laryngoscope?**

Ans. Laryngoscopes are devices that have been designed to visualize the interior of the larynx including the vocal cords so as to aid endotracheal intubation.

History: In 1895, Alfred Kirstein developed a direct vision laryngoscope. It was popularized by Sir Robert Mackintosh and Sir Ivan Magill in the early 1940.

Q. What are the uses of a laryngoscope?

Ans.

- For intubation
- Foreign body removal
- Upper airway lesion biopsy
- Visualization of vocal cord and larynx
- For insertion of nasogastric tube.

Q. Can you briefly describe the parts of a laryngoscope?

Ans. Its parts consist of the following:

Handle: Its function is to apply suitable leverage to the blade. It has a rough surface for traction and usually houses the batteries for the light source. Handles are designed to accept blades that have a light bulb with a metallic contact. When the handle and the blade are engaged in the operating position, it completes an electrical circuit. Handles are available in several sizes. A narrow handle is useful while intubating a small patient. Short handles are useful in patients in whom the chest and/or breasts contact the handle during use, or if cricoid pressure is being given.

Fitting: The connection point between the handle and the blade is called the fitting. The hook on the fitting is most commonly used. Another type of fitting employs a pin which fits through holes in the blade and the handle. When ready to use, most blades form a right angle with the handle, but they may also form acute (Guedel and Bennett) or obtuse (Polio, Bowen-Jackson) angles. An adaptor may be fitted between the handle and blade to allow the angle to be altered. The Patil-Syracuse handle can be positioned and locked in four different positions.

Blade: It is a rigid component that is inserted into the mouth. It helps to elevate the lower jaw and tongue. The blade further consists of the following (**Fig. 7**):

- **Base:** It is the part that attaches to the handle. It has a slot for engaging the hinge pin of the handle.
- **Heel:** The proximal end of the base is called the heel.
- **Spatula (tongue):** It is the main shaft. It serves to compress and manipulate the soft tissues and the lower jaw so that a direct line of vision to the larynx is achieved. The long axis of the tongue may be straight or curved in part or all of its length.
- **Flange:** It is parallel to the tongue and is connected to it by the web. It serves to guide instrumentation and deflect interfering tissues. The flange determines the cross-sectional shape of the blade.
- **Tip (beak):** It contacts either the epiglottis or vallecula and directly or indirectly elevates the epiglottis. It is usually blunt and thickened to decrease trauma.

The light source is a bulb screwed on to the blade. The electrical conduit runs at the back of the blade. An electrical connection is made when the blade is opened and the light comes on and the laryngoscope is ready for use. Most of the time the light source is near the tip of the blade, but in some blades, it is in the base of the blade.

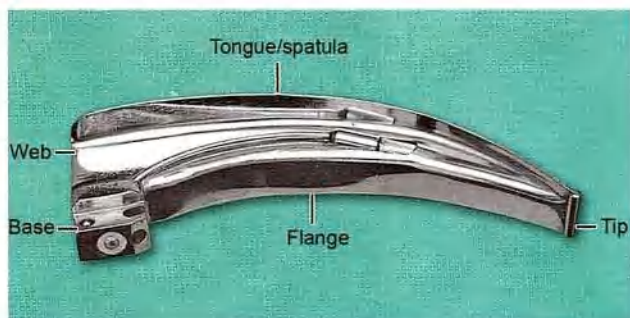


Fig. 7: Macintosh blade.

Q. What have been the recent advances in the laryngoscope design?

Ans. Some designs place the bulb in the handle and the light is transmitted to the blade by the means of fiberoptics. The fiberoptic bundle may be an integral part of the blade or it may be detachable. The light intensity is greater than that provided by the regular light bulb. As there is no bulb or electrical contact in the blade, cleaning and sterilization are easier and the laryngoscopes are more reliable. Even if the light is left on for the extended period of time, the blade stays cool. Also, there is no danger of the bulb getting detached and acting as a foreign body. Rechargeable nickel-cadmium batteries are becoming popular as power source for laryngoscopes. Xenon gas-filled bulbs have brighter light than conventional tungsten-type bulbs; however, they require power from alkaline batteries to work efficiently.

Q. Which is the most commonly used laryngoscope? Is there a standard laryngoscope for all patients?

Ans. The Macintosh laryngoscope is most commonly used. The spatula has a smooth gentle curve that extends from the base to the tip. There is a flange at the left to push the tongue out of the way. In cross-section, the spatula and the flange form a reverse "Z." Its tip is blunt and hence causes lesser trauma to the tissues. It has four sizes, i.e., infant, child, medium adult, and large adult. Its blades are interchangeable. Left-sided Macintosh is also available for left-handed anesthetists and oral or facial lesions on the right side. This is also helpful in intubating in right lateral position.

There is no standard laryngoscope for all patients. Depending on the indication, various laryngoscope handles and blades can be used. There are different laryngoscopes for adults and pediatric patients. Also, the laryngoscope blades come in different sizes. The line of sight direct laryngoscopy can be limited by various anatomical obstacles such as maxillary incisors, size of tongue, retrognathia, and anterior larynx.

Miller blade is a straight blade laryngoscope available in adult and children sizes. It is designed to lift the epiglottis directly and is particularly useful if a large, floppy, or irregularly shaped epiglottis is encountered during laryngoscopy. These blades have smaller displacement volumes and are favored in patients with smaller displacement space (e.g., children or patients with micrognathia, prominent upper incisors, or short mental-hyoid distance). Modified straight blades include the Wisconsin and the Guedel, which have higher flange heights compared with the traditional Miller blade.

Q. What are the unique features of laryngoscopes used for difficult intubations?

Ans. McCoy laryngoscope: It is based on the standard Macintosh blade with the addition of an adjustable tip that is operated by a lever on the handle. It is inserted in the normal way and if the larynx is not clearly visualized, then the tip can be flexed so that it elevates the epiglottis. Less force is required to bring the larynx into view. The point on the blade, which acts as a fulcrum, is moved further into the pharynx, thereby eliminating any inadvertent contact with the upper teeth. It is suitable both for routine use and in cases of difficult intubation. This laryngoscope may use either a traditional bulb in the blade or a fiberoptic light source. It is most useful in cases of Cormack-Lehane grade 3, where it can lift the epiglottis and aid in visualization of glottis. In cases where laryngoscopy has to be done in neutral head position, this blade can improve glottis view.

Bullard laryngoscope: The blades are designed so that varying degrees of jaw protrusion and upper neck extension will bring either a part or the entire larynx into direct view. It has a curved blade which is designed to pass behind the tongue and lift it away from the posterior pharyngeal wall without any neck extension. The fiberoptic viewing channel and light source incorporated in the blade helps in the visualization of the larynx by indirect laryngoscopy.

The blade may be passed anterior to the epiglottis as with a Macintosh blade or posterior to it as with a straight blade. A suitable endotracheal tube is preloaded on a wire fitted to the side of the device and runs parallel to the blade. The fiberoptics allow the visualization of the tip of the guide as it has a slight bend. It also allows the visualization of the tube as it enters the larynx. Intubation is easier with a reinforced tube. This laryngoscope is useful in patients with limited neck movement and/or restricted mouth opening. Both adult and pediatric sizes are available.

Q. Which laryngoscope would be useful in obese patients?

Ans. Polio blade: It is also a modification of the Macintosh blade. The blade is offset from the handle at an obtuse angle to allow intubation of patients in iron lung respirators or body jackets, when the screen is in place after induction of anesthesia, obese patients, patients with large breasts, and patients with kyphosis with severe barrel chest deformity, short neck, or restricted neck mobility.

Disadvantage: Little force can be applied and control is minimal.

Q. What is an Oxiport Macintosh?

Ans. Oxiport Macintosh is a conventional Macintosh blade with a tube added to deliver oxygen.

Q. What are the uses of a left-handed Macintosh laryngoscope blade?

Ans. The left-handed Macintosh laryngoscope blade has a flange on the opposite side from the usual Macintosh blade. This blade is useful for abnormalities of the right side of the face (e.g., cleft palate) or oropharynx, left-handed persons, intubating in the right lateral position, and positioning a tracheal tube directly on the left side of the mouth.

Q. Which laryngoscope is useful in patients with limited mouth opening?

Ans. Bizarri-Giuffrida blade: It is a modified Macintosh. The flange is removed except for a small part that encases the light bulb so that the space required to insert the blade in the mouth is minimum. This was done in an attempt to limit damage to the upper teeth. It was designed particularly for patients with limited mouth opening, prominent incisors, receding mandible, short thick neck, or anterior larynx.

Q. Can you describe the laryngoscopy technique?

Ans. Laryngoscopy consists of several steps: Positioning the head, opening the mouth, inserting the blade, identifying the epiglottis, and finally viewing the larynx.

Position for Laryngoscopy (Two Positions)

Optimal position for most patients is flexion of lower cervical spine (35°) and extension of the head (15°) at atlanto-occipital joint (sniffing position). The lower part of the cervical spine can be maintained in a position of flexion by means of a small pillow under the head. Extension at the atlanto-occipital joint is achieved by pressure on top of the head and/or upward traction on upper teeth or gums by the laryngoscopist's hand. The goal is alignment of three axes, i.e., the oropharyngeal, pharyngeal, and laryngeal as the anatomical basis for the position.

Head and neck in full extension (Chevalier Jackson position): In children, it may be unnecessary to flex the lower cervical vertebrae, and in neonates it may be necessary to elevate the shoulders.

Direct laryngoscopy can be thought of as progressive visualization of intraoral and pharyngeal soft tissue structures as the laryngoscope blade is advanced and landmarks are exposed. The laryngoscope blade functions as both a retractor and an illuminator during this process. The laryngoscope is held in the left hand. The fingers of the right hand are used to open the mouth and spread the lips apart. The blade is inserted at the right side of the mouth. This reduces the likelihood of damage to the incisors and helps push the tongue to the left. The blade is advanced on the side of the tongue toward the right tonsillar fossa, so that the tongue lies on the left side of the blade. The right hand keeps the lips from getting caught between the teeth or gums and the blade. When the right tonsillar fossa is visualized, the tip of the blade is moved toward the midline. The blade is then advanced behind the base of the tongue, elevating it, until the epiglottis comes into view. The jaw and the tongue are lifted away from the posterior pharyngeal wall to expose the larynx and epiglottis. The direction of the lifting force should be along the axis of the laryngoscope handle (i.e., parallel to the handle) to avoid using the laryngoscope blade as a lever and potentially damaging upper teeth or other structures. This maneuver creates a space that is larger on the right hand side for insertion of the endotracheal tube. There are two methods for elevating the epiglottis depending whether a straight or curved blade is being used.

Straight blade: After exposure of the epiglottis, the blade is withdrawn slightly to scoop under the epiglottis and lift it forward. If the larynx is not visualized downward external pressure on the larynx may be useful. If the laryngoscope is advanced too far, it will override the slanting glottis and

enter the esophagus. If it is then withdrawn too far, the tip of the epiglottis will be released and will flop down over the glottis. Straight blades are useful in infants and children. It is also useful in adults with long floppy epiglottis.

Curved blade: When the epiglottis is seen, the blade is advanced till the tip fits into the vallecula. Traction is then applied along the handle, at right angles to the blade to carry the base of the tongue and the epiglottis forward. If the glottis is not seen, then external pressure on the larynx may be helpful. The handle should not be pulled backward as it will cause the tip to push the larynx upward and out of sight and could cause damage to the teeth and gums.

Alternative techniques are the paraglossal, left molar, and retromolar approaches that have been suggested for difficult direct laryngoscopies.

In obese patients as the head extension is limited, head elevation with alignment of the patient's sternal notch and external auditory meatus in a horizontal plane may be superior to the sniffing position. This is also called the rapid airway management positioner (RAMP) position.

Q. What is Cormack-Lehane grading of glottis view?

Ans. Cormack-Lehane classification (1984) is the original, and most widely used classification of laryngeal view. Higher Cormack-Lehane views (grades III and IV) are associated with difficult intubation (**Fig. 8 and Table 1**).

TABLE 1: Cormack-Lehane classification grades.

Grade 1	Most of the glottis is visible
Grade 2a	Posterior part of cords visible
Grade 2b	Only arytenoids visible
Grade 3a	Visible epiglottis "moveable"
Grade 3b	Epiglottis visible with difficulty
Grade 4	No part of larynx visible

Q. What is BURP (Backward Upward Rightward Pressure) maneuver and optimum external laryngeal manipulation (OELM)?

Ans. In the BURP technique, posterior laryngeal pressure against the cervical vertebrae is applied with superior and (rightward) lateral pressure. When the view of glottis is poor, maneuvers such as OELM, applied either by an assistant or with the laryngoscopist's right hand, may improve the view.

Q. Can you enumerate the complications of laryngoscopy?

Ans.

- Damage to the teeth, gums, and dental prosthesis
- Bruising and laceration of lips and tongue, abrasion and laceration of hard and soft palate, the posterior wall of pharynx, epiglottis and valleculae, and blunt trauma or lacerations of larynx and/or esophagus. The risk of trauma and bruising is higher with the straight blade. Lingual nerve may be injured
- Injury to cervical spinal cord
- Changes in the cardiac rhythm and blood pressure are common during laryngoscopy
- Swallowing or aspiration of a foreign body (e.g., bulb).

Burns: Malpositioning of blade on handle can produce a short circuit, which leads to rapid heating of handle.

Laryngoscope malfunction: Most common is failure of the light to illuminate. This may be a result of defective power source, defective lamp, faulty socket, or poor contact between the blade and the handle:

- Laryngeal spasm
- Vagal stimulation.

Q. How are laryngoscopes sterilized?

Ans. Blades are cleaned with soap and water followed by spirit or chlorhexidine swabbing to disinfect it. Blades can be autoclaved without the light carrier.

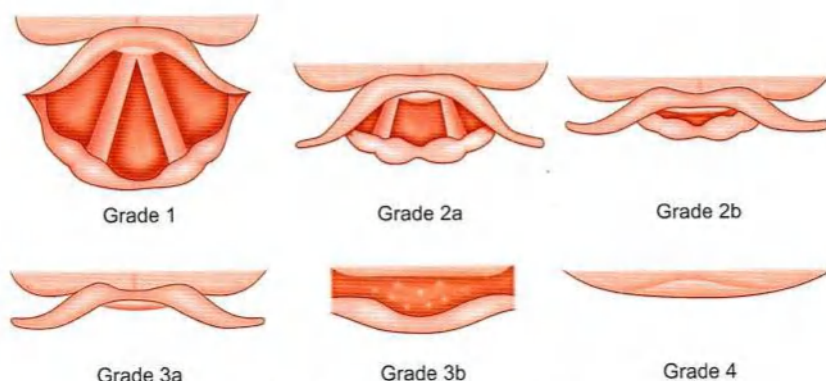


Fig. 8: Cormack-Lehane grading.

■ NONREBREATHING VALVES

Q. Define nonrebreathing valves (NRVs).

Ans. Nonrebreathing valves ensure unidirectional flow of gases. The various components are arranged to ensure that during the inspiratory phase, gas flows only into the patient port and during the expiratory phase, the exhaled gas escapes through the expiratory port without mixing with the fresh gas stored in the bag.

Q. What are the properties of an ideal NRV?

Ans. An ideal NRV should:

- Have minimal dead space
- Have minimal resistance
- Have no forward/backward leak
- Have light weight
- Be transparent
- Be easy to clean and sterilize
- Be non-noisy
- Be nonsticky
- Be compact and inexpensive
- Have minimal opening pressure.

Q. What are the advantages of NRVs?

Ans. The advantages of NRVs are as follows:

- Prevent rebreathing if valve is functioning properly.
- Inspired concentration of gases and vapors can be controlled as there is no mixing.
- Help dissipation of heat and water vapor.
- Due to low resistance, they can be used for spontaneous and controlled respiration.
- They can be used to measure the minute volume. The sum of the fresh gas flows on the flowmeter will be equal to the minute volume if the fresh gas flows are adjusted such that the reservoir bag is three-fourth inflated at the end of expiration.
- Eliminate hazards of carbon dioxide absorption (heat retention, inhalation of irritant dust, and exhausted soda lime).
- Compact, light-weight, and mobile.
- Inexpensive.
- Dead space is minimal in a properly functioning valve.
- While using NRVs, the feel of the bag is a good indicator of compliance as the only distensible parts of the system are the patient's lungs and the reservoir bag.

Q. What are the disadvantages of NRVs?

Ans. The disadvantages of NRVs are as follows:

- Wastage of expensive anesthetic gases as relatively large flows is needed for adults. This increases the cost and causes theater pollution; there is a hazard of explosions when flammable agents are used.
- There is considerable loss of heat and humidity from the patient.

- They are bulky and hence may cause the tube to kink or be displaced downward.
- They cause theater pollution as scavenging is difficult (except in Laerdal valve).
- Some of the valves are noisy and sticky when wet.
- If the expiratory valve becomes stuck, then high pressures may be built up leading to barotrauma.
- Cleaning and sterilization may be difficult.
- During spontaneous respiration, frequent adjustments in the fresh gas flows may be required to match the patient's minute volume.
- When used in adult patients, the higher gas flows required may cause increased resistance.
- In some valves, the dead space can be relatively high for pediatric patients.
- There is no standardization of the valves.
- If the expiratory valve sticks during spontaneous respiration, then it can cause air dilution.
- In some valves, both hands are required for assisted/controlled ventilation.
- In some valves, if the bag release is delayed, then it can cause rebreathing due to backward leak.
- During controlled ventilation in some valves, the gas can escape past the expiratory valve during the initial part of inspiration. This is known as forward leak which can reduce the tidal volume delivered to the patient.
- The feel of the bag is different as compared to other systems.

Q. How would you classify NRVs?

Ans. Nonrebreathing valves can be classified depending on the type of respiration and depending on the position of the valve.

Sykes classification depending on the type of respiration: From the functional point of view, the main difference between spontaneous and controlled ventilation is the pressure inside the valve during inspiration. During controlled ventilation, there is positive pressure inside the valve and during spontaneous ventilation, the patient exerts a negative pressure which is transmitted to the inside of the valve.

Valves designed for spontaneous respiration, e.g., Stephen Slater valve.

During inspiration, the negative pressure exerted by the patient closes the exhalation port. During exhalation, the pressure in the valve increases and gas escapes through the exhalation port. If this valve is used for controlled or assisted ventilation, it is necessary to close the exhalation port with a finger during inspiration.

Valves designed for controlled respiration, e.g., Ambu resuscitation valve.

In this type of valve, a pressure increase opens the inlet and closes the exhalation port. If the patient is allowed to breathe spontaneously, he will inspire room air through the exhalation port. It cannot be useful for general anesthesia.

Valves designed for both spontaneous and controlled ventilation, e.g., Ruben valve, Ambu E, Ambu E2, Ambu Hesse, Laerdal valve, and Lewis Leigh valve.

In this type of valve during inspiration, the exhalation port is closed and the inlet opens with either controlled or spontaneous respirations. During expiration, the exhalation port opens to allow free exhalation and inspiratory port closes to prevent rebreathing. Only dual purpose valves are used in anesthesia.

Depending on the Position

- *Positional valve* works on gravity and hence must be horizontal.
- *Nonpositional valves* are closed by elastic tension of rubber or by a spring and may be used in any position, e.g., flap valve, fish mouth valve, and disc type.

Q. How would you describe a nonrebreathing valve?

Ans. While describing an NRV, first classify it. Then give a brief description of the valve and explain how it functions. What is the dead space and resistance of the valve? Then talk about its advantages and disadvantages. Finally describe how the valve is sterilized.

Q. Give a brief description of the Ruben valve. How does it function?

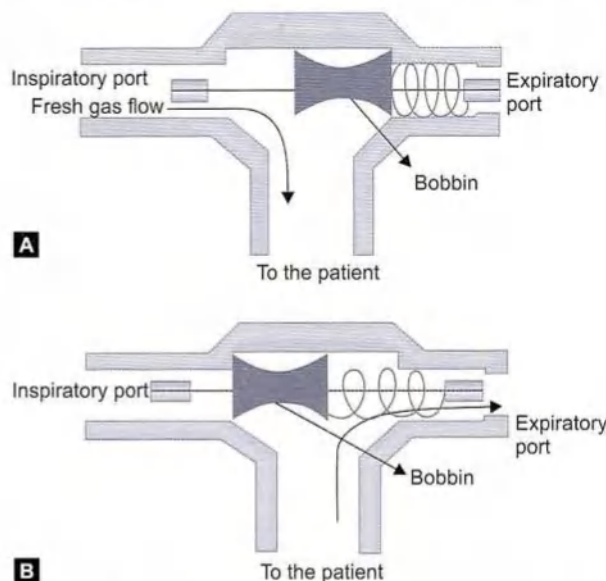
Ans. Classification:

- *Ruben resuscitation valve:* It is used only for controlled respiration.
- Ruben mark 2 dual purpose valve.

Construction: It has a clear plastic body with metal fittings. It is a multicolored valve in which blue color is inlet, gold color is outlet, and red color end is patient's end. Ruben valve has a spring-loaded bobbin within the valve housing. The exhalation port is perforated and has a disc-shaped valve.

Working: In resting position, the spring holds the bobbin away from the expiratory port and against the inspiratory port allowing unhindered exhalation to occur via patient port. When the bag is squeezed, the bobbin is forced across the valve housing to occlude the expiratory port. The inspiratory port is now connected to the patient port and the inspiratory gases enter the patient's lungs without leaking out through the expiratory port. In spontaneous respiration, the patient draws air from the inspiratory limb, thus forcing the bobbin to move and occlude the expiratory port. When the pressure at the inlet drops, the force of the spring pushes the piston against the inlet. During exhalation, the pressure of the exhaled gases causes the disc valve to open. The gas then passes to the atmosphere.

When the valve is to be used with resuscitation equipment, the exhalation valve is not present. This allows the patient to inspire from the atmosphere (**Figs. 9A and B**).



Figs. 9A and B: Ruben valve: (A) Inspiratory phase; (B) Expiratory phase.

Dead space: 9 mL

Resistance: Inspiratory—0.8 cm H₂O at 25 L/min and expiratory—1 cm H₂O at 25 L/min.

Disadvantages:

- Noisy
- Cleaning is difficult
- Presence of backward leak (some exhaled gases may go back to reservoir bag). With fast bag release, the backward leak is 10–12% of tidal volume; with slow bag release, it is 32–44%; and the backward leak could be as high as 76% of tidal volume in patients with increased resistance and decreased compliance
- If valve sticks in inspiration, high pressure may be formed leading to barotrauma
- If the valve is not correctly assembled, then it can lead to increased resistance and rebreathing
- If exposed to heat, it may deteriorate.

Ruben Valve

Care and cleaning: Valves can be cleaned by flushing the channels with soap and water. It can also be gas sterilized.

Q. What are the types of Ambu valves available? What are the differences between them?

Ans. Ambu valves are of two types which are as follows:

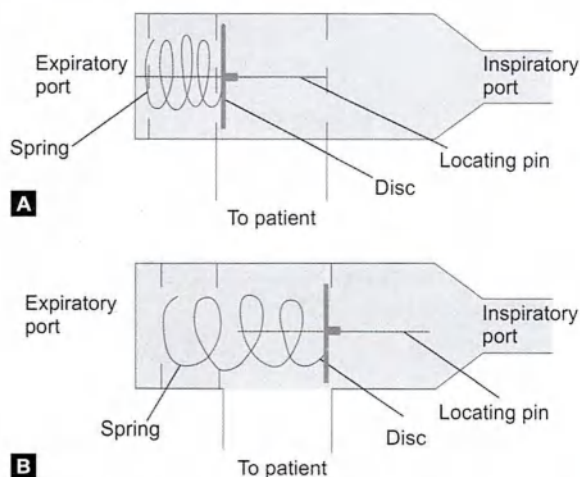
1. For controlled ventilation, e.g., Ambu valve
2. For dual purpose, e.g., Ambu E, Ambu Hesse, and Ambu E2.

Ambu valve: This is commonly used with Ambu resuscitation bag.

Classification: Controlled respiration.

Construction: Valve is made of metal or plastic material. It has a yellow plastic disc, which is the movable part. It is held in place by a spring. A locating pin centers the disc.

Function: During controlled respiration, when the bag is squeezed, the force of the gas pushes the disc and closes the exhalation port, thus allowing the patient's lung to be inflated. During exhalation, the pressure on the inlet side of the disc falls and the spring pushes the disc toward the inlet. The exhaled gases pass from the patient out through the exhalation port. During spontaneous ventilation, the interior of disc does not move and the patient inhales atmospheric air (**Figs. 10A and B**).



Figs. 10A and B: Ambu valve. (A) Inhalation; (B) Exhalation.

Care and cleaning: It can be disassembled for cleaning. When reassembling, it is essential to insert the guide pin through the appropriate channel. The valve should be checked for competence after reassembling.

Ambu E valve: In this, the unidirectional flow of gases is controlled by two labial flap valves.

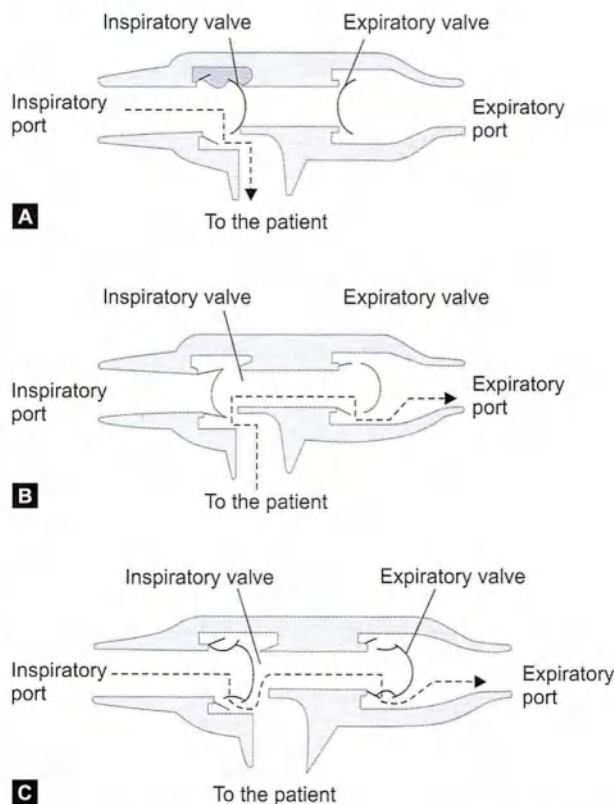
Classification: Spontaneous or controlled respiration.

Construction: It is made up of transparent plastic. The inlet connection is blue colored. It has two labial flaps one for inhalation and one for exhalation.

Working: During spontaneous respiration, the negative pressure created by the patient during inspiration closes the valve at the expiratory port, while the fresh gases flow in through the inspiratory port. During expiration, the valve at the inspiratory port closes and the valve at the expiratory port opens so as to allow the expired gases to be voided to

the atmosphere. The valve at the expiratory port prevents inhalation of the downstream gases during spontaneous ventilation. This downstream gas may be air when the valve is used as a resuscitator, but it could be expired gases if used in a circle breathing system. During expiratory pause, excess gases may pass outside straight—away as both valves open partially.

When Ambu E is used for controlled ventilation, initially very high pressures are required to produce sufficient movement of the valve at the inspiratory port in order to completely seal the expiratory port. At lower inspiratory flow rates, this valve is inefficient because there is incomplete sealing of the expiratory port which allows some of the inspiratory gas to pass straight across the valve resulting in lower than expected tidal volumes (**Figs. 11A to C**).



Figs. 11A to C: Ambu E valve. (A) Inspiration; (B) Expiration; (C) Expiratory pause.

Dead space: 10 mL.

Resistance: Inspiratory resistance is 0.6–2.1 cm H₂O at 5–40 L/min and expiratory resistance is 0.6–2.5 cm H₂O at 5–40 L/min.

Advantage: It prevents excessive build-up of pressure.

Disadvantage: It cannot be used with resuscitators that do not produce initial high surge of gas required to produce an

effective seal. The backward leak is 9% of the tidal volume with slow bag release and 1.8% with fast bag release.

Ambu E2 valve: It contains single unidirectional valve. It has no valve at the expiratory end.

Classification: Spontaneous and controlled respiration.

Working: The negative pressure created by the patient during spontaneous inspiration opens the inspiratory port. It also draws air from the expiratory port. The result is that a mixture of fresh gas and air is inhaled. During controlled or assisted respiration, the positive pressure from bag squeezing causes the inspiratory valve to occlude the expiratory port, so that fresh gases pass to the patient. During expiration, the inspiratory port closes and exhaled gases pass through the expiratory port to the atmosphere.

Dead space: 10 mL.

Backward leak: It is 9% of tidal volume with slow bag release and 1.8% with rapid release.

Care and cleaning: Parts can be cleaned with soap and water. The parts may also be boiled, autoclaved, or gas-sterilized.

Ambu Hesse valve: It is similar to Ambu E valve, but is slightly larger.

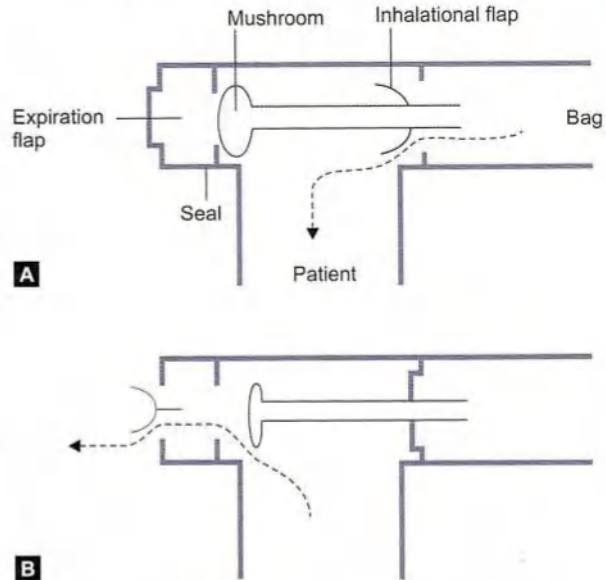
Classification: Spontaneous and controlled respiration.

Construction: This valve overcomes the potential problem of leakage across the Ambu E valve at low flow rates. The valve mechanism has three components, i.e., an inspiratory leaf, an expiratory leaf, and a mushroom valve.

Working: During controlled ventilation, manual compression of the self-inflating bag causes a small increase in pressure within the bag. This causes the elastic mushroom valve to expand and totally occlude the expiratory port providing a complete seal. If the bag is compressed further, it opens the inspiratory leaf valve, forcing gas from the bag into the inspiratory port, thus providing inspiratory flow. At the beginning of exhalation phase, the self-inflating bag starts to reexpand. The reduced pressure within causes the mushroom valve to collapse and the inspiratory leaf valve to close, sealing the bag off from the main valve. The exhaled patient gases then leave the system through the expiratory port via the expiratory leaf valve (**Figs. 12A and B**).

If the valve is used for spontaneous breathing, the negative pressure produced by the patient during inspiration causes the expiratory leaf valve to close, sealing the expiratory port. This ensures that the inspired gas is drawn from the bag and not from the ambient air through the expiratory port. During expiration, the valve functions the same as during controlled ventilation.

Dead space: 14 mL.



Figs. 12A and B: Working of Ambu-Hesse: (A) Inspiration; (B) Expiration.

Resistance: Inspiration resistance is 0.2–0.7 cm H₂O for flows from 5–40 L/min and expiration resistance is 0.2–0.9 cm H₂O for flows from 5–40 L/min.

Backward leak: It is 7.3% of the tidal volume with slow bag release and 0.9% with rapid release.

Care and cleaning: The valves can be disassembled for cleaning. The parts may be boiled, autoclaved, or gas sterilized.

Q. Which NRV is most commonly used and why?

Ans. The Laerdal valve is most commonly used. It is light weight, has a transparent body, smallest dead space, and minimal resistance to breathing. It can be used for both spontaneous and controlled ventilation. It can be easily dismantled and sterilized.

Q. How does a Laerdal valve work?

Ans. Classification: Spontaneous or controlled ventilation.

Construction: It has a clear plastic body and contains a rubber inspiratory fish mouth valve with a circular flange. There is a circular exhalation flap valve which occludes the exhalation port. It is available in three sizes, i.e., adult, child, and infant. All sizes have 23 mm external diameter expiratory port and 22 mm external diameter/15 mm internal diameter inspiratory port to minimize misconnections.

Working: In spontaneous respiration, the fish mouth valve opens and allows the gas to pass to the patient during inspiration. Simultaneously, the expiratory port is sealed as the outer disc portion of the valve is pushed against the apertures in the valve body. The exhalation flap prevents the room air from being inhaled through the exhalation ports.

In the expiratory phase, positive pressure from the elastic recoil of the patient's lungs causes the fish mouth valve to close preventing rebreathing into the bag. It also lifts the outer disc-shaped portion of the expiratory apertures, thus allowing exhaled gases to escape.

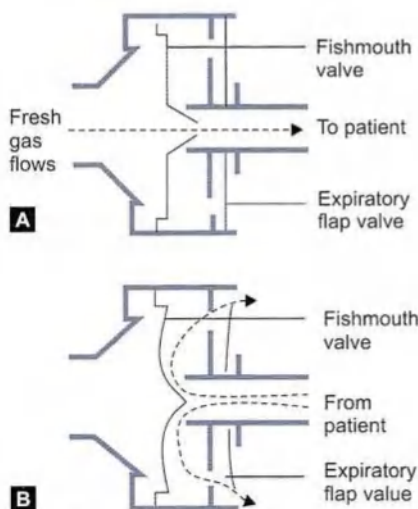
During controlled ventilation, the pressure at the inlet opens the fish mouth valve and pushes the disc portion of the flap valve against the exhalation ports during inspiration. This allows the fresh gases to pass to the patient and prevents its loss through the exhalation ports. During exhalation, the valve functions the same as with spontaneous respirations (**Figs. 13A and B**).

The child and infant models have overpressure safety valves built into the inspiratory port and are set to blow off at a pressure of 45 cm H₂O. If these pressures need to be exceeded, the safety valve can be overridden by finger pressure or a lock clip over the valve.

Dead space: 7 mL.

Resistance: Inspiration resistance is 0.32–1.82 cm H₂O for flows from 4.7–44.6 L/min. Expiration resistance is 0.16–2.83 cm H₂O for flows from 5.0–54.4 L/min.

Care and cleaning: The valve can be disassembled to clean or repair broken parts. It can be cleaned with chemical solutions, boiling, or autoclaving.



Figs. 13A and B: Laerdal valve: (A) Inspiration; (B) Expiration.

Q. Give a brief description and working of the Lewis Leigh valve.

Ans. Classification: Spontaneous or controlled ventilation.

Construction: It has a clear plastic body with an exhalation chimney which may be rotated 90°. There is a disc valve at the top of the chimney and an exhalation valve seat at the bottom of the chimney. The chimney is rotated clockwise for controlled respiration and counter-clockwise for spontaneous respiration. Rotation of the chimney causes changes in the position of the valves which in turn facilitates respiration. This valve is a modification of the Digby Leigh valve which had a simple disc type exhalation valve instead of the chimney. The exhalation valve had to be closed with a finger during controlled or assisted ventilation.

Working-controlled ventilation: During inspiration, the gas flows to the patient as the flap valve moves away from its seat on the body and closes over the base of the chimney. At the end of inspiration, when the pressure in the system returns to atmospheric, the flap valve returns to its resting position over the fresh gas inlet. As the patient exhales, the flap valve seats on the ridge and the exhaled gases pass up the chimney lifting the disc valve. Gas then flows to the patient. When the chimney is rotated 90° counterclockwise, the gas passes through the flap valve and travels to the patient during inspiration. The disc valve in the chimney prevents air from being drawn into the valve during spontaneous ventilation. During exhalation, the exhaled gases pass into the chimney. Any excess gas can then leak through the valve to atmosphere thereby preventing build-up of pressure inside the valve.

Resistance: The resistance is 0.1 cm H₂O for flows from 3–5 L/min.

Backward leak: With fast bag release, the backward leak is 8–11% of tidal volume and with slow bag release, the backward leak is 10–29% of tidal volume.

Care and cleaning: The valve can be gas-sterilized.

Q. Which valve has the smallest dead space and which valve has the largest dead space?

Ans. Laerdal valve has the smallest dead space of about 7 mL and Ambu Hesse valve has the largest dead space of about 14 mL (**Table 2**).

TABLE 2: Comparison chart for nonrebreathing valves.

	Ambu	Ambu E	Ambu E2	Ambu Hesse	Laerdal	Ruben
Housing material	Polycarbonate and polysulfonate	Polycarbonate and polysulfonate	Polycarbonate and polysulfonate	Polycarbonate and polysulfonate	Polycarbonate and polysulfonate	Plastic
Fittings material	Silicone	Silicone	Silicone	Silicone	Silicone	Metal fittings

Contd...

Contd...

	<i>Ambu</i>	<i>Ambu E</i>	<i>Ambu E2</i>	<i>Ambu Hesse</i>	<i>Laerdal</i>	<i>Ruben</i>
Components of the valve	Spring, pin, and disc	Two labial flaps	Single labial flap	Mushroom and two flap valves	Fish mouth, circular flange, and expiratory flap	Spring-loaded bobbin and flap
Dead space	10 mL	10 mL	10 mL	14 mL	7 mL	9 mL
Inspiratory resistance (cm H ₂ O)	0.6–2	0.6–2	0.6–2	0.2–0.7	0.32–1	0.9
Expiratory resistance (cm H ₂ O)	0.6–2	0.6–2	0.6–2	0.2–0.9	0.16–2.83	1
Salient features	Not suitable for spontaneous respiration	High forward leak	Not suitable for spontaneous respiration	Low resistance	Least dead space	High backward leak

Q. Which are the valves with backward leak?

Ans. Ambu E, Ambu Hesse, Ambu E2, Lewis Leigh, and Ruben valves (**Table 2**).

■ SUGGESTED READING

1. Collins SR. Direct and indirect laryngoscopy: equipment and techniques. *Respir Care*. 2014;59(6):850-62.
2. Davey AJ, Diba A. *Ward's Anaesthetic Equipment*, 5th edition. Philadelphia: Saunders; 2005.
3. Dorsch JA. *Understanding Anesthesia Equipment*, 1st edition.
4. Dorsch JA, Dorsch SE. *Understanding Anesthesia Equipment*, 5th edition. Philadelphia: Lippincott Williams & Wilkins; 2008.

Electrocardiography

Reshma Ambulkar, Swapnil Parab, Bindiya Salunke

Q. Describe the various leads used to obtain an electrocardiogram (ECG).

Ans. A 12-lead electrocardiography (ECG) provides a complete picture of the electrical activity of the heart by recording information from 12 different views of the heart.

Limb leads: There are six limb leads (leads I, II, III, aVR, aVF, and aVL). Leads I, II, and III require a positive electrode and a negative electrode and are hence called bipolar leads. Leads aVR, aVF, and aVL are augmented unipolar leads as they require only a positive electrode. In these leads, the small wave forms are enhanced or augmented. The last letter refers to the position of the lead.

Chest leads: V_1 , V_2 , V_3 , V_4 , V_5 , and V_6 are unipolar leads as only positive electrode is required for generating a waveform, the negative electrode being the center of the heart.

Q. Enumerate leads and their corresponding regions of the heart.

Ans.

- **Leads I and aVL:** Left side of the heart
 - **aVF, lead II, and lead III:** Inferior wall of the heart
 - **V_2 , V_3 and V_4 :** Anterior surface of the heart
 - **V_1 and V_2 :** Septal area
 - **V_5 and V_6 :** Apical surface
 - **Mirror image changes in leads V_1 – V_4 :** Posterior surface
- Table 1** below provides information about arterial blood supply to different regions of the heart.

TABLE 1: Arterial blood supply in different regions of the heart.

Region of heart	Arterial blood supply
Anteroseptal	Left anterior descending artery
Anteroapical	Left anterior descending artery
Anterolateral	Circumflex artery

Contd...

Contd...

Region of heart	Arterial blood supply
Inferior	Right coronary artery
Posterior	Right coronary artery

Sinoatrial (SA) node and atrioventricular (AV) node are supplied by the right coronary artery in >80% of individuals. Patients with inferior wall myocardial infarction (MI) have an increased incidence of developing a complete heart block.

Q. Describe the ECG paper.

Ans. The paper consists of squares—large and small. 5 small squares make 1 large square. 300 large squares or 1,500 small squares are covered in a minute of ECG recording (with paper speed of 25 mm/s). This makes 1 small square = 0.04 second, 1 large square = 0.2 second, and 5 large squares = 1 second. Vertical axis represents the voltage and 1 small square in the vertical axis is equal to 0.1 mV at normal settings.

Q. How would you describe a normal ECG by a systematic approach?

Ans. Standardized sequence of steps to analyze the ECG is as follows (**Fig. 1**):

- Rate
- Rhythm
- Axis
- P wave
- PR interval
- QRS complex
- ST segment
- T wave
- QT interval
- U wave
- Conclusion.

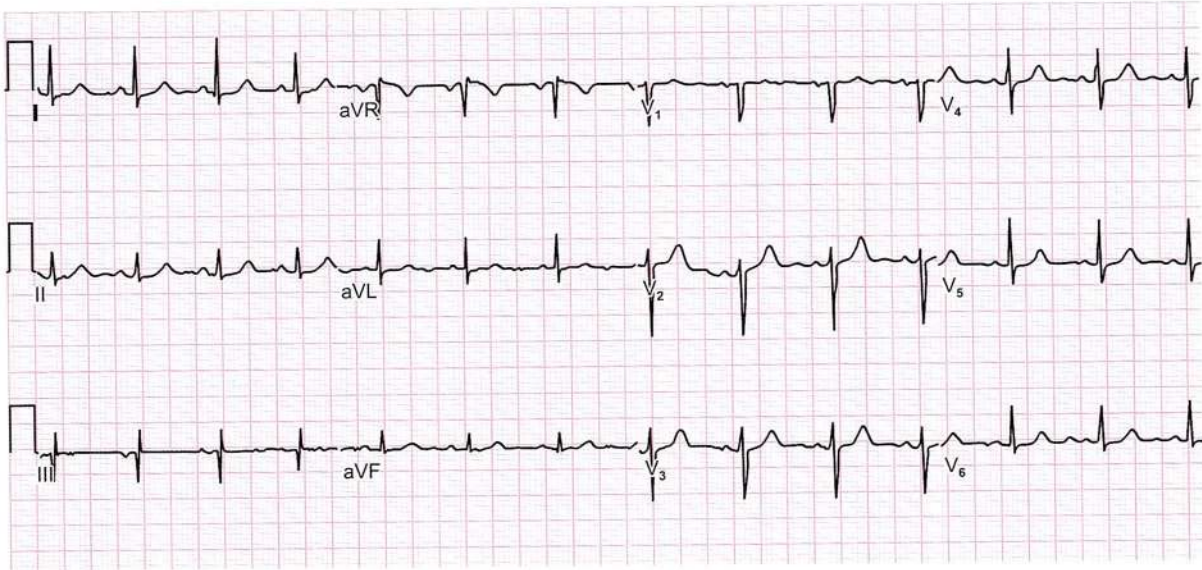


Fig. 1: Normal ECG.

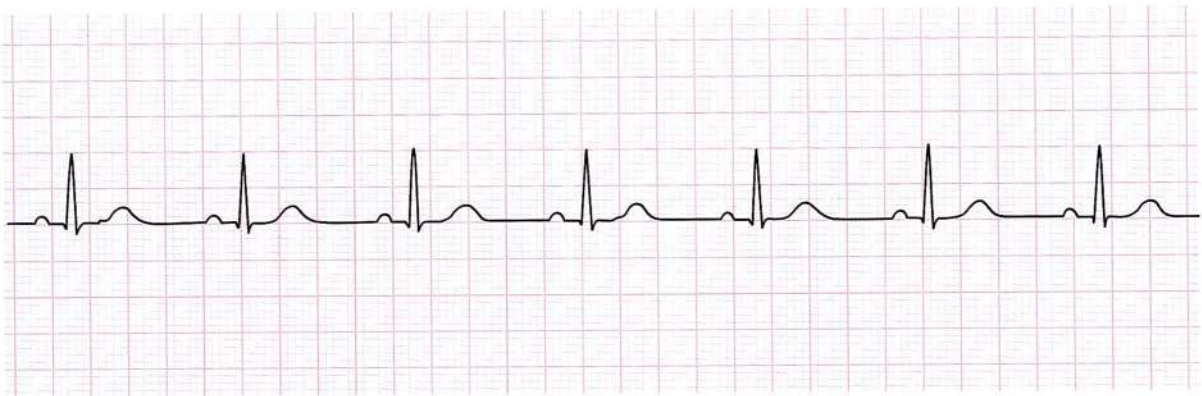


Fig. 2: Calculation of heart rate in patient with regular rhythm.

Q. How do you calculate heart rate from ECG?*Ans.*

- When rhythm is regular, one can calculate rate by either:
 - Dividing 1,500 by the number of small squares between one R-R interval or
 - Dividing 300 by number of large squares between one R-R interval.
- When rhythm is irregular, one can calculate rate by:
 - Counting the number of R-R intervals in 3 seconds (15 large squares) and multiplying by 20.

Rule of 10: Calculate the number of R waves in an ECG strip (normally, one standard ECG strip is a record of 10 seconds) and multiply it by 6.

Example (Fig. 2): Heart rate = $300/4 = 75$ beats/min

Example (Fig. 3): Heart rate = total number of R waves in one ECG strip $\times 6 = 14 \times 6 = 84$

Q. How do you calculate the axis?*Ans.*

Calculation of axis of heart (Fig. 4):

- Normal range: -30° to $+90^\circ$
- Left axis deviation: -30° to -90°
- Right axis deviation: $+90^\circ$ to $+150^\circ$.

Calculate the net QRS deflection for leads I and aVF (you can also take other two pairs, i.e., II and aVL or III and aVR but stick to these pairs only, do not mix them) by adding up the number of small squares that correspond to the height of the R wave (positive deflection) and subtracting the number of small squares that correspond to the depth of the Q and S waves (negative deflection). Now plot these points on the X-axis and Y-axis, draw perpendiculars from these points, and mark a point where these perpendiculars meet. Pass a line from the center of circle to this point which will give you the axis.

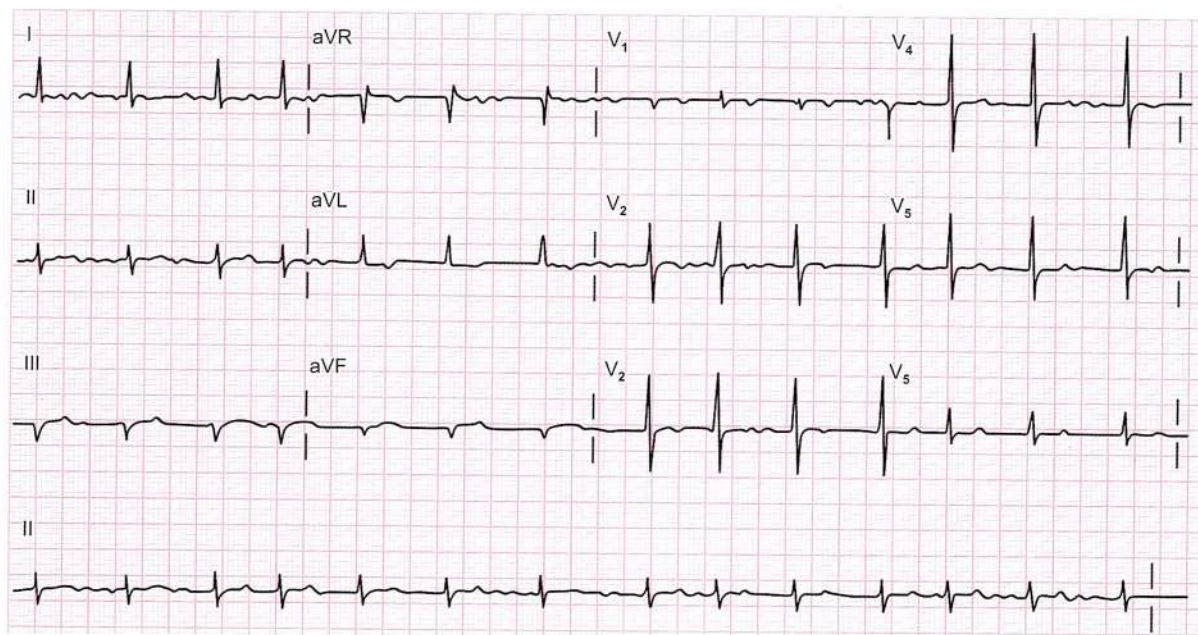


Fig. 3: Calculation of heart rate in a patient with irregular rhythm.

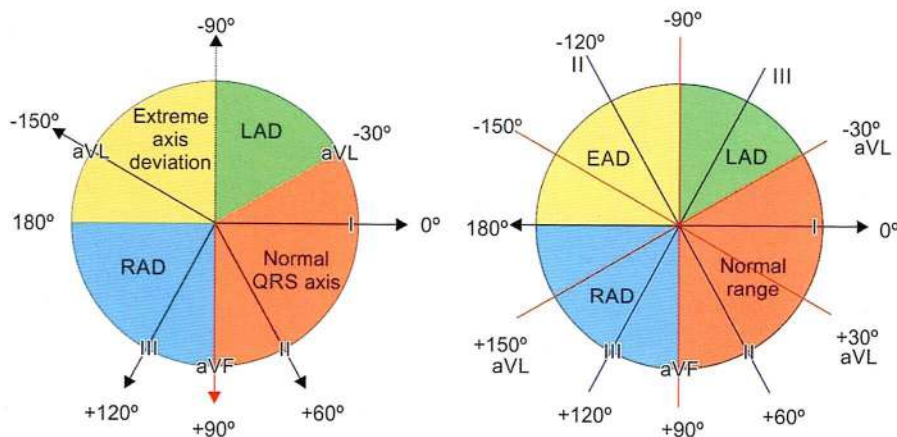


Fig. 4: Calculation of the axis of the heart.

Other way of calculating axis: Examine six limb leads—I, II, III, aVR, aVL, and aVF.

- Locate the lead (X) with equiphasic deflection (the lead with equal forces in the positive direction and negative direction)
- The axis would run perpendicular to that lead (X)
- Since there are two perpendiculars to each isoelectric lead, choose the perpendicular that best fits the direction of the other ECG leads.

Q. Explain the waves and complexes seen in the ECG.

Ans. Table 2 summarizes the waves and complexes seen in ECG.

TABLE 2: Waves and complexes seen in ECG.

Waves/Complexes		Duration
P wave	It represents the spread of electrical activation (depolarization) through the atrial myocardium	3 mm in height (0.3 mV) or 3 mm horizontally (0.12 second)
PR interval	PR interval is measured from the beginning of the P wave to the beginning of the QRS complex. Represents the time taken by the electrical impulse to travel from the sinus node through the AV node to the bundle of His	0.12–0.2 second

Contd...

Contd...

Waves/ Complexes		Duration
QRS complex	QRS complex represents ventricular depolarization	0.08–0.12 second Voltage varies according to the leads, position of heart, and abnormality
T wave	T wave represents the repolarization of the ventricles	0.12–0.16 second
U wave	U wave represents the repolarization of the papillary muscles or Purkinje fibers	
QT interval	QT interval is measured from the beginning of the QRS complex to the end of the T wave. The QT interval represents the time for both ventricular depolarization and repolarization (ventricular action potential). QTc— independent of heart rate	QT: 0.2–0.4 second QTc: <0.44 second
ST segment	It is the time at which the entire ventricle is depolarized and roughly corresponds to the plateau phase of the ventricular action potential	0.08–0.12 second
J point	J point is the junction between the QRS complex and the ST segment. It is the point from where the degree of ST elevation or depression is measured	

Q. What does the P wave signify? What are the abnormalities seen with P wave?

Ans. It represents the spread of electrical activation (depolarization) through the atrial myocardium. Normally, it does not exceed 3 mm in height (0.3 mV) or 3 mm horizontally (0.12 second).

Abnormalities:

- **Absent:**
 - Atrial fibrillation
 - SA block
 - Nodal rhythm
- **Inverted:**
 - Dextrocardia
 - Incorrect electrode placement
 - Junctional rhythm
- **Wide and notched:**
 - P mitrale—left atrial enlargement (LAE)
- **Tall and peaked:**
 - P pulmonale—in right atrial enlargement (RAE).

Q. What abnormalities are seen in the T wave?

Ans.

- **T wave inversion:**
 - Q wave and non-Q wave MI

- Myocardial ischemia
- Subacute MI or old pericarditis
- Myocarditis
- Myocardial contusion (from trauma)
- Central nervous system (CNS) disease causing long QT interval
- **Tall T wave:**
 - Hyperkalemia
 - Postwall MI
- **Flat T wave:**
 - Emphysema
 - Pericardial effusion
 - Myxedema
 - Hypokalemia
 - Hypocalcemia.

Q. When do you see prominent U waves?

Ans.

- Hypokalemia
- Hypercalcemia
- Thyrotoxicosis
- **Drugs:** Digitalis, epinephrine, and antiarrhythmics
- Intracranial hemorrhage.

An inverted U wave can be found in myocardial ischemia or left ventricular volume overload (**Fig. 5**).

Q. What is the significance of long QT syndrome and what are its causes?

Ans. Long QT syndrome (QTc > 0.47 second in males and >0.48 second in females) (**Fig. 6**).

Significance: Increased vulnerability to malignant ventricular arrhythmias, syncope, and sudden death.

Causes:

- Drugs (many antiarrhythmics, tricyclics, and phenothiazines)
- Electrolyte abnormalities (decreased K⁺ or Ca⁺⁺ or Mg⁺⁺)
- CNS disease (especially subarachnoid hemorrhage, stroke, and trauma)
- Hereditary long QT syndrome (e.g., Romano-Ward syndrome)
- Coronary heart disease (post-MI patients).

Q. What is corrected QT interval?

Ans. The QT interval is dependent on the heart rate (the faster the heart rate, the shorter the QT interval) and needs to be adjusted to improve the detection of patients at increased risk of ventricular arrhythmia. This is calculated by using Bazett's formula: $QTc = QT/\sqrt{RR}$.

Q. How can one differentiate between Wolff-Parkinson-White (WPW) syndrome and Lown-Ganong-Levine (LGL) syndrome?

Ans. WPW syndrome: An accessory pathway (called the "Kent" bundle) connects the right atrium to the right

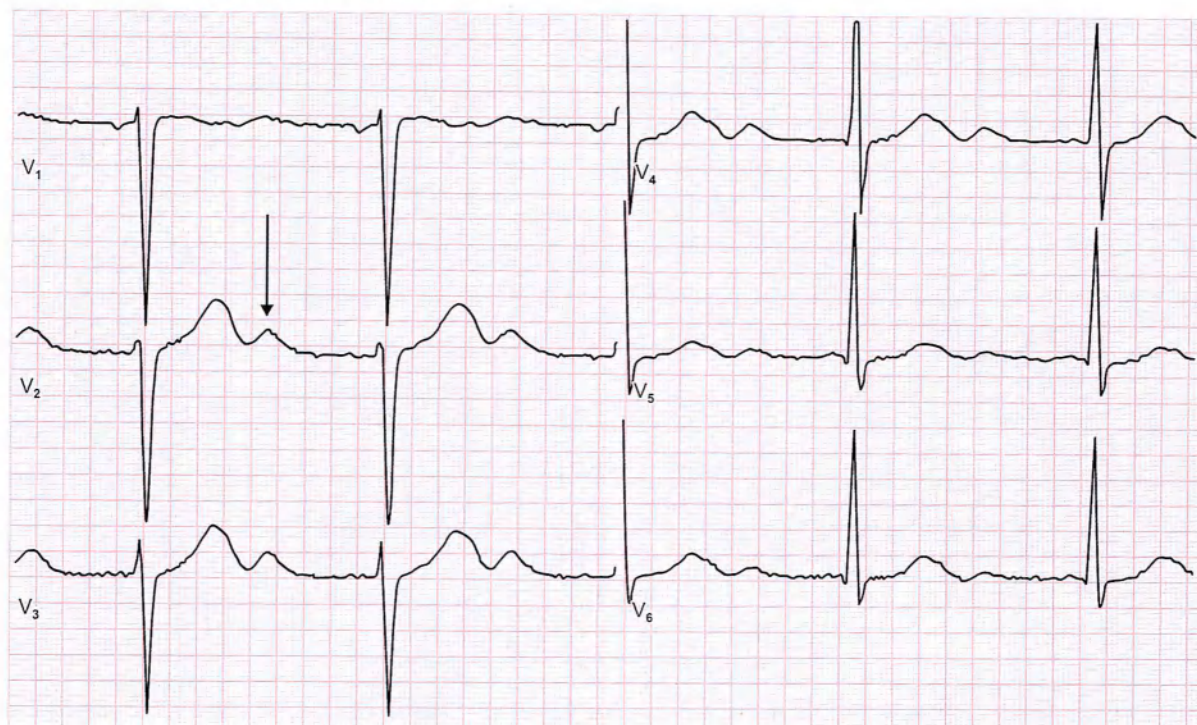


Fig. 5: ECG showing U wave.

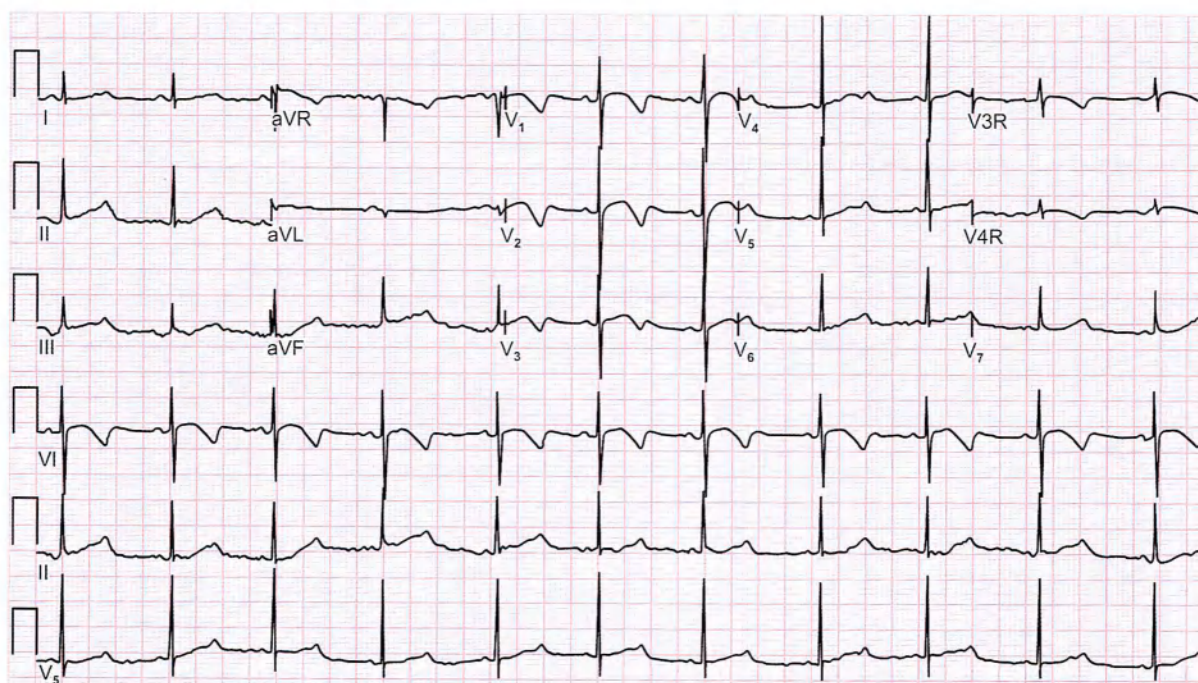


Fig. 6: ECG with prolonged QT interval.

ventricle and the left atrium to the left ventricle, and this causes an early activation of the ventricles (delta wave) and a short PR interval. The features are as follows (Fig. 7):

- Short PR interval (<0.12 second)
- Initial slurring of QRS complex (delta wave) representing early ventricular activation through normal ventricular muscle in the region of the accessory pathway
- Prolonged QRS duration (usually >0.12 second)

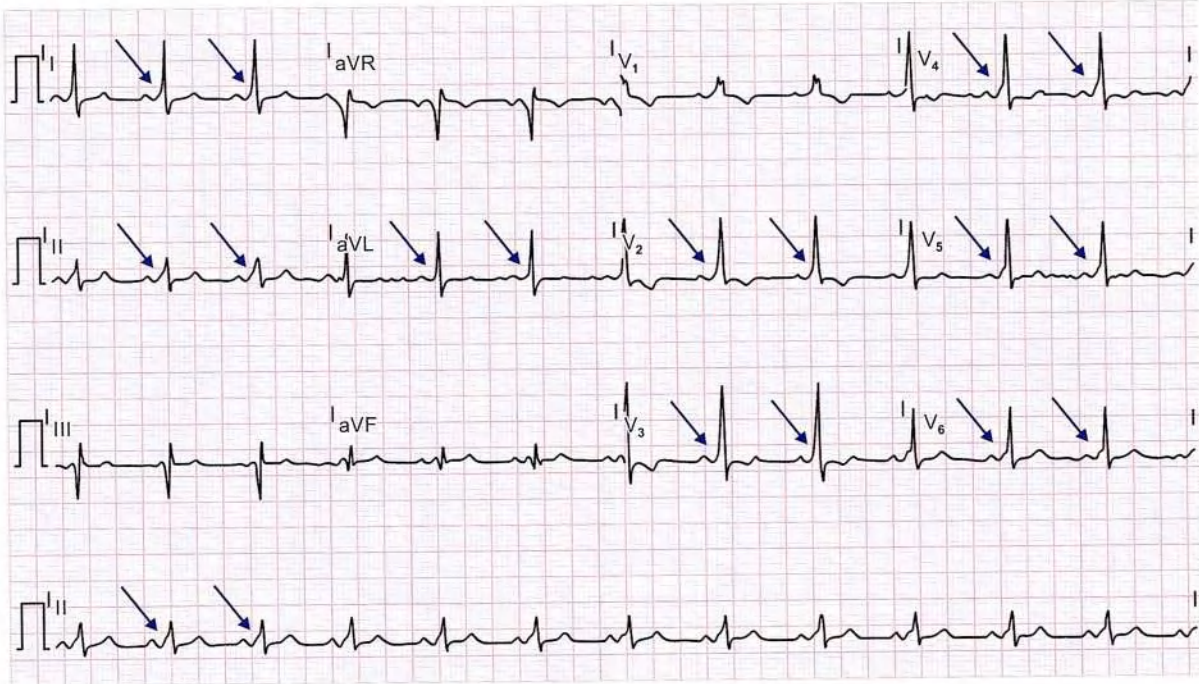


Fig. 7: WPW syndrome. Note delta waves shown by arrows.

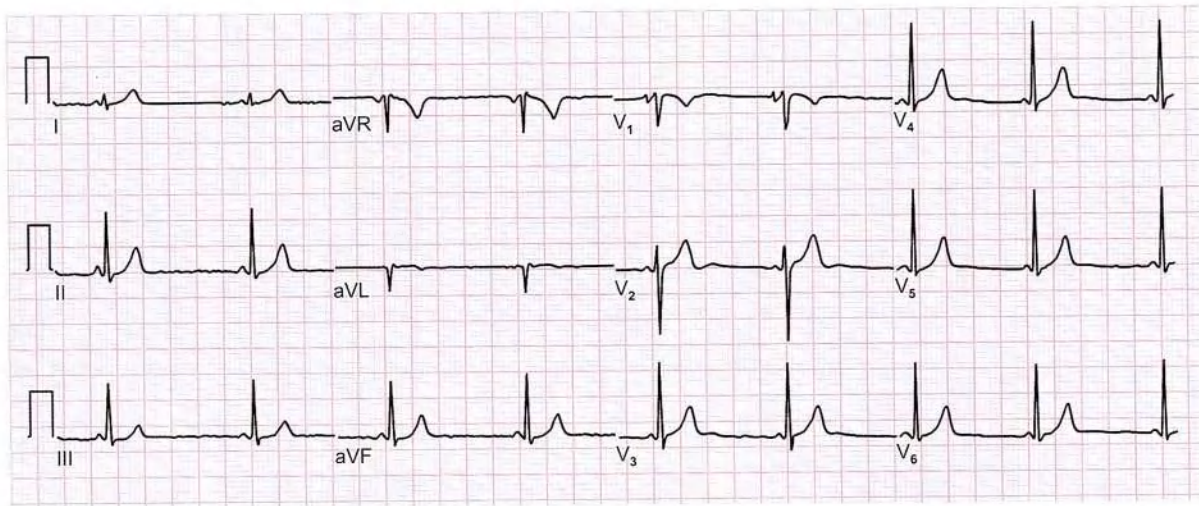


Fig. 8: ECG with LGL syndrome. Note short PR interval and absence of delta waves.

- Secondary ST-T changes due to the altered ventricular activation sequence.

LGL syndrome: An AV nodal bypass tract exists in the His bundle, and this permits early activation of the ventricles (short PR interval) without a delta wave because the ventricular activation sequence is normal (**Fig. 8**).

Q. List the differential diagnosis of ST segment depression.

Ans. Normal variants or artifacts:

- Pseudo-ST depression (wandering baseline due to poor skin-electrode contact)
- Physiologic J-junctional depression with sinus tachycardia.

Ischemic heart disease:

- Non Q-wave MI
- Reciprocal changes in acute Q-wave MI (e.g., ST depression in leads I and aVL with acute inferior MI).

Nonischemic causes of ST depression:

- Right ventricular hypertrophy (RVH) (right precordial leads) or left ventricular hypertrophy (LVH) (left precordial leads, I, aVL)—called as strain pattern
- Digoxin effect on ECG

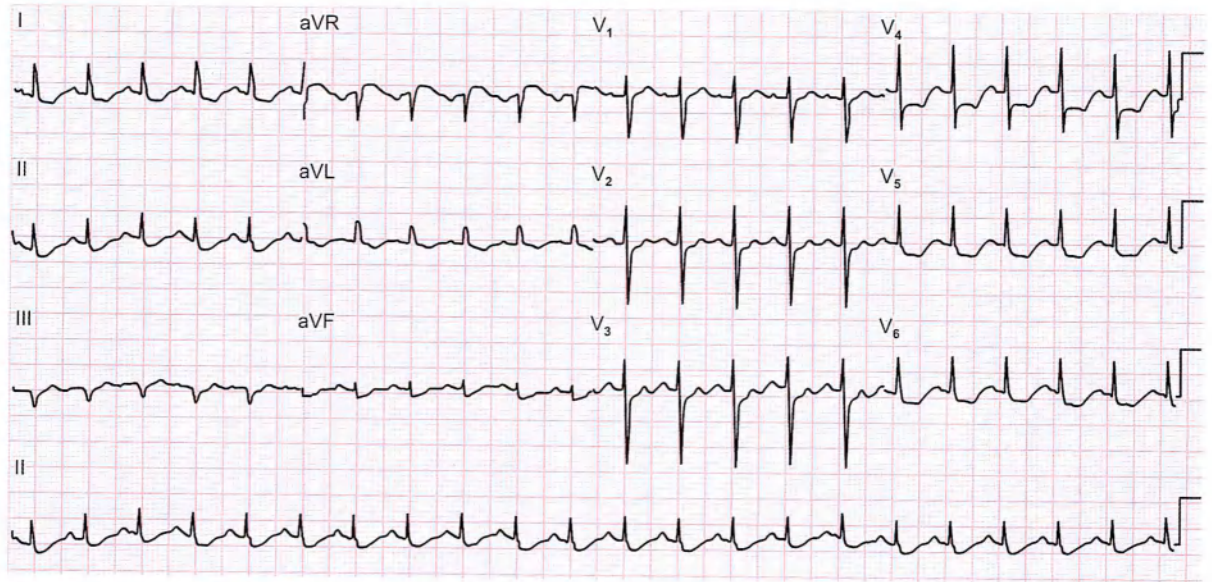


Fig. 9: ECG with ST segment depression in anterolateral leads.

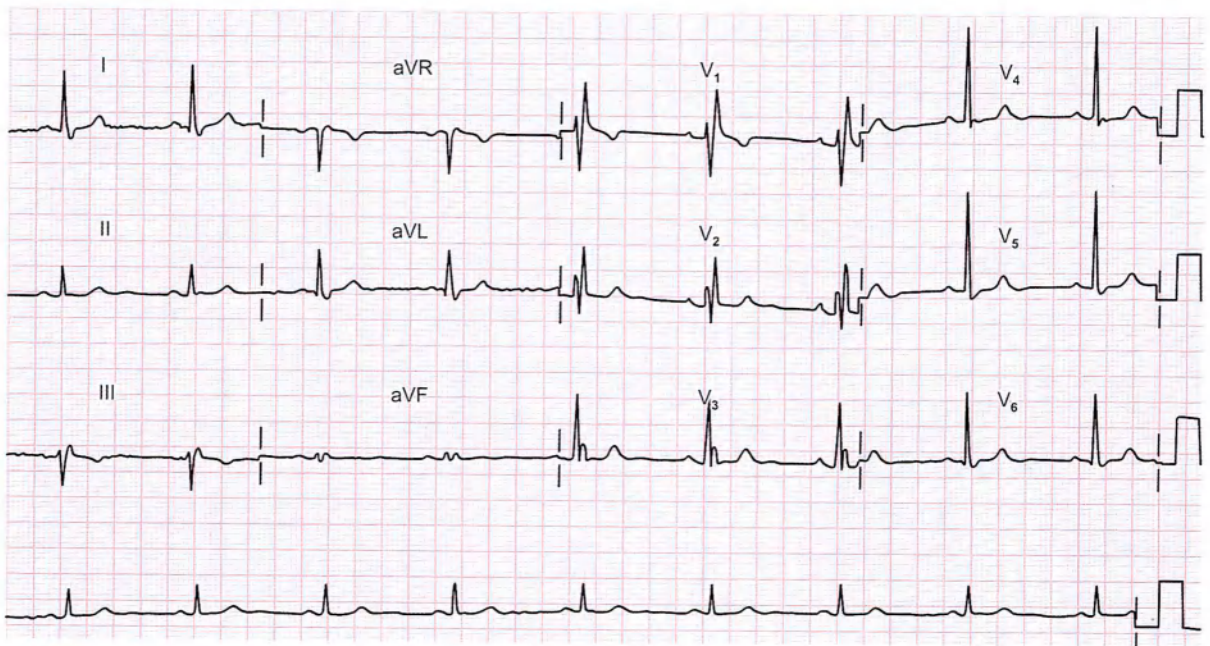


Fig. 10: ECG with right bundle branch block.

- Hypokalemia
- Mitral valve prolapse
- Subdural hematoma. **Figure 9** shows the ST segment depression in anterolateral leads.

Q. What are the causes of right bundle branch block (RBBB) and criteria for diagnosis?

Ans. Causes of RBBB:

- Normal individuals
 - Transient in pulmonary embolism
 - Coronary artery disease
 - Atrial septal defect.
- Criteria for diagnosis:*
- Right axis deviation
 - QRS duration > 0.12 second
 - Secondary R wave is seen in V₁ and V₂ resulting in M-shaped QRS complex
 - Deep, slurred S waves in left precordial leads (typically V₄, V₅, and V₆). **Figure 10** shows the electrocardiogram with right bundle branch block.

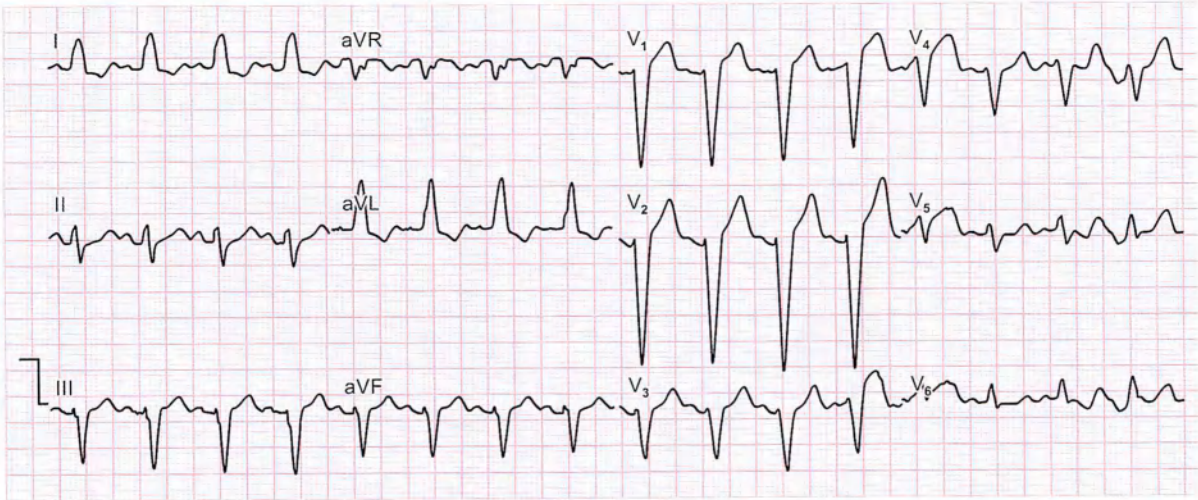


Fig. 11: ECG with left bundle branch block.

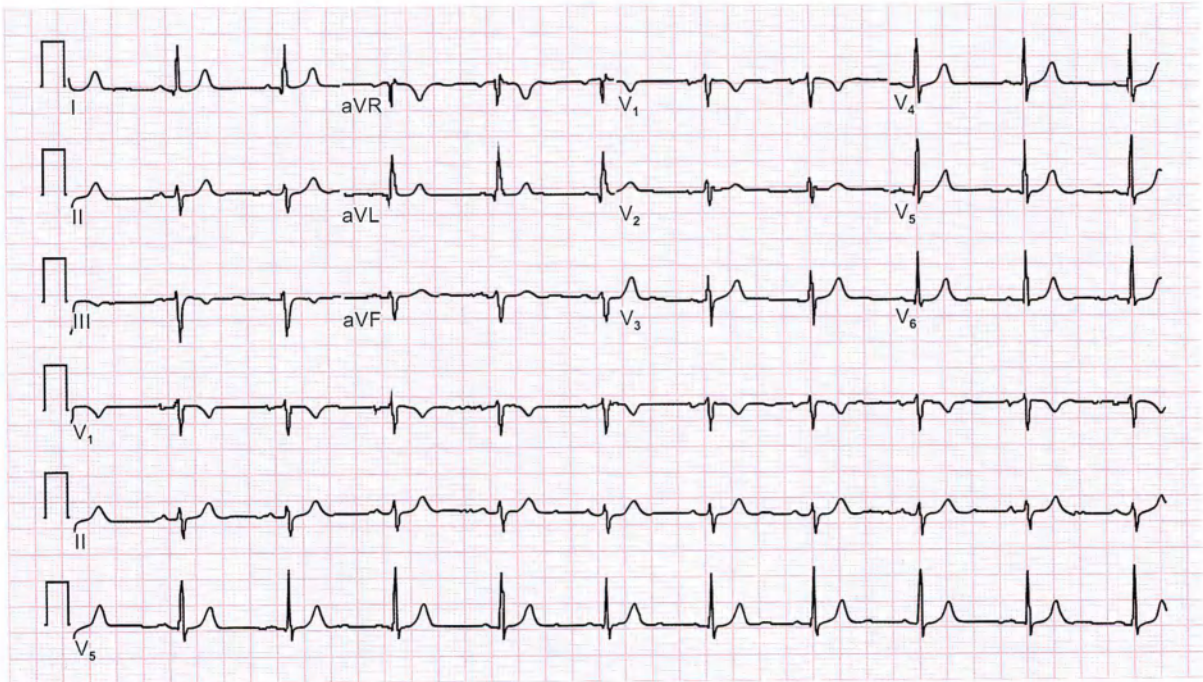


Fig. 12: ECG with left anterior hemiblock.

Q. How do you diagnose left bundle branch block (LBBB) on ECG?

Ans. Diagnosis of LBBB (Fig. 11):

- Left axis deviation
- QRS duration > 0.12 second
- M pattern QRS complexes in V₄, V₅, and V₆.

Causes of LBBB:

- Ischemic heart disease
- Myocarditis
- Hypertension

- Cardiomyopathy
- Aortic valve disease.

Q. How would you diagnose left anterior hemiblock (LAHB) and left posterior hemiblock (LPHB)?

Ans. Hemiblock: Conduction is delayed in one of the divisions of left bundle branch (LBB).

Left anterior hemiblock: Conduction is delayed in the anterosuperior division of LBB so activation is through the posteroinferior division (Fig. 12).

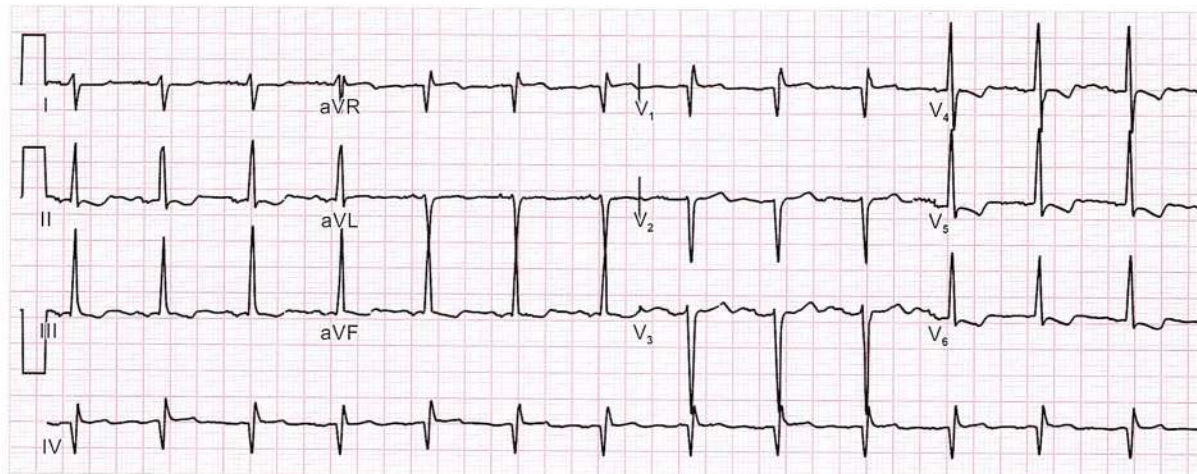


Fig. 13: ECG with left posterior hemiblock.

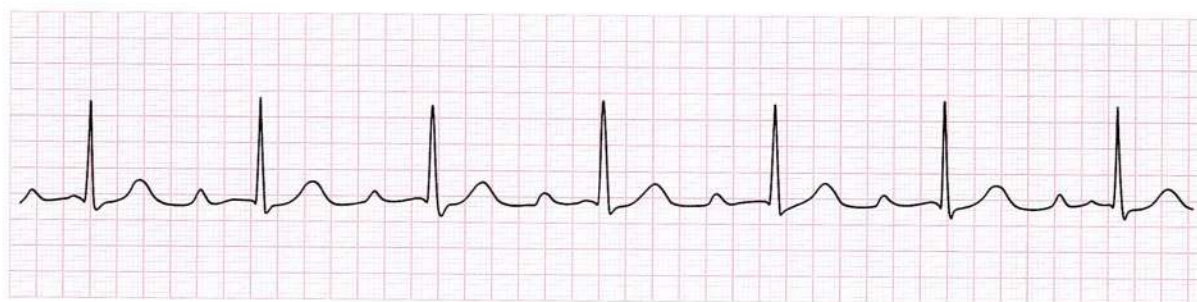


Fig. 14: ECG with first-degree heart block.

Criteria for LAHB: Left axis deviation with QRS axis more negative than -30° and deep S waves in inferior limb leads (leads II, III, and aVF).

Left posterior hemiblock: Conduction is delayed in the posteroinferior division of LBB (Fig. 13).

Criteria for LPHB: Right axis deviation and tall R wave in all inferior limb leads (leads II, III, and aVF).

Q. What are the diagnostic criteria for first-degree heart block?

Ans. First-degree heart block is a delay in conduction of impulse passing through the AV node (Fig. 14).

Criteria:

- Prolongation of PR interval >0.2 second
- Rhythm is regular
- All beats conducted from the atria to the ventricle.

Causes:

- Idiopathic
- Coronary artery disease
- Digitalis toxicity
- **Drugs:** β -blockers.

Q. How do you differentiate between Mobitz type I heart block (Wenckebach phenomenon) and Mobitz type II heart block?

Ans. Mobitz type I (Wenckebach phenomenon) (Fig. 15): PR interval increases gradually until a P wave fails to conduct to the ventricles.

Mobitz type II (Fig. 16):

- PR interval constant
- Intermittent block in conduction of the P wave to the ventricles
- Lesion usually in the bundle of His.

Causes of Mobitz types I and II:

- Acute rheumatic carditis
- Coronary heart disease
- **Drugs:** Digitalis and quinidine toxicity.

Q. How do you diagnose complete heart block on ECG?

Ans.

- **Characterized by complete interruption of AV conduction:** AV dissociation (Fig. 17).
- P wave bear no relationship to QRS complexes—P walking through QRS.

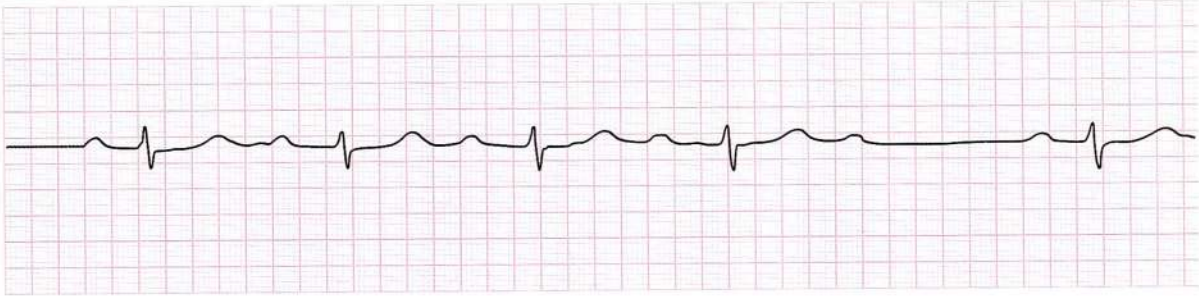


Fig. 15: ECG with Mobitz type I block.

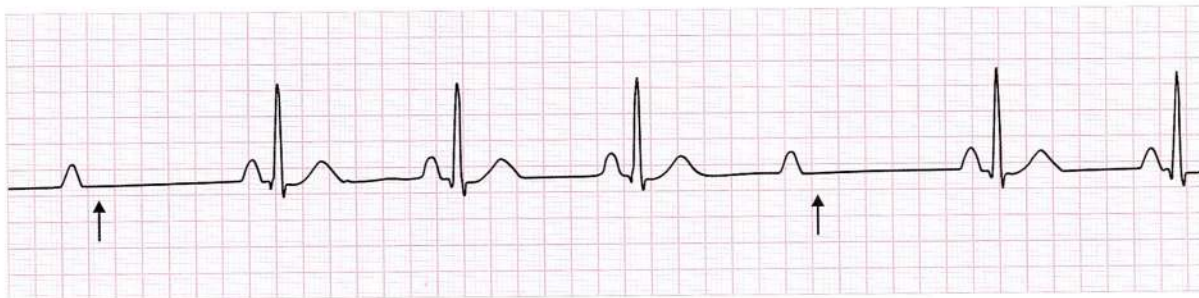


Fig. 16: ECG with Mobitz type II block.

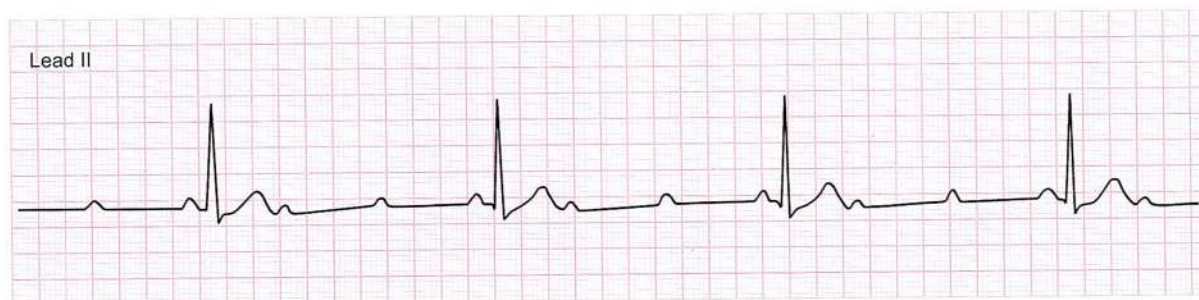


Fig. 17: ECG with complete heart block.

- P waves coming at regular interval in different rates and QRS coming at regular interval at different rates
- A slow ventricular rate.

Q. What are the indications for insertion of transvenous pacemaker?

Ans. Refer to the chapter on Anesthesia for a patient with pacemaker.

Q. What are bifascicular and trifascicular blocks?

Ans. Bifascicular block: More commonly the combination of RBBB with left anterior fascicular block (manifests as LAD) and less commonly RBBB with left posterior fascicular block (manifests as RAD). Examples of RBBB + LAHB are shown in **Figure 18**.

Trifascicular block: Combination of bifascicular block with first-degree heart block.

RHYTHM ABNORMALITIES

Q. What is sinus arrhythmia?

Ans. It is a naturally occurring variation in the heart rate that occurs during a breathing cycle, causing an increase in the heart rate during inspiration and decrease during expiration.

It is due to nucleus ambiguus (center in the medulla oblongata), which increases the parasympathetic nervous system input to the heart via the vagus nerve. Upon expiration, the cells in the nucleus ambiguus are activated and the heart rate is slowed down and during inspiration, there are inhibitory signals to the nucleus ambiguus.

It is more marked in the younger age group.

Normal PQRST complexes with alternating long and short PP interval.

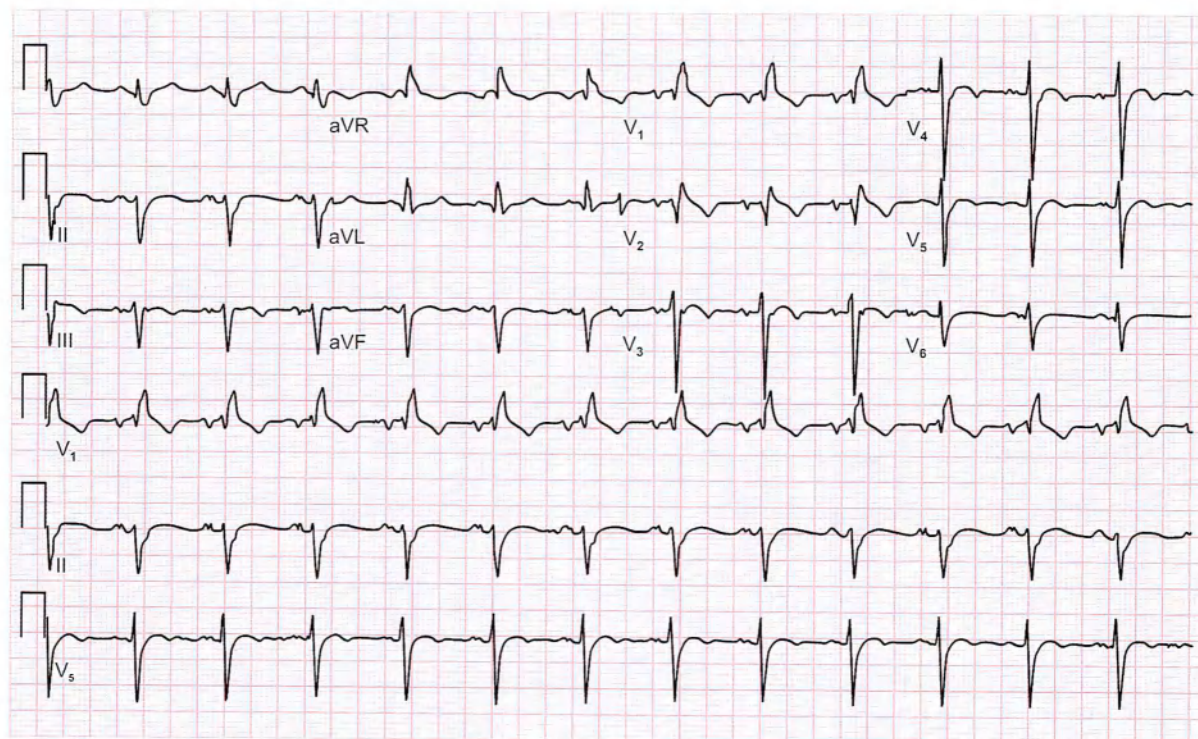


Fig. 18: ECG with right bundle branch block plus left anterior hemiblock.

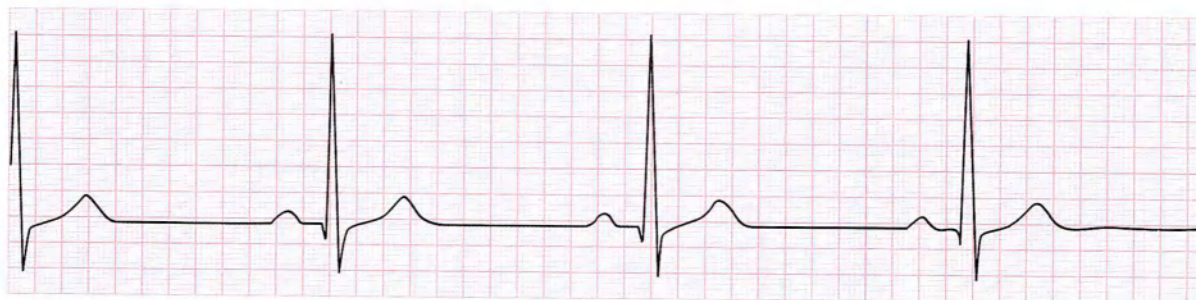


Fig. 19: Sinus bradycardia.

Accentuated: Carotid sinus compression

Abolished: Exercise and atropine

Q. Define sinus bradycardia and sinus tachycardia?

Ans. Sinus bradycardia: When the SA node discharges at a rate slower than 60/min (**Fig. 19**).

Sinus tachycardia: When the SA node discharges at a rate faster than 100/min.

Both are associated with normal PQRST complexes.

Q. What are the causes of sinus bradycardia?

Ans. Physiological:

- Sleep
- Athletes
- Starvation.

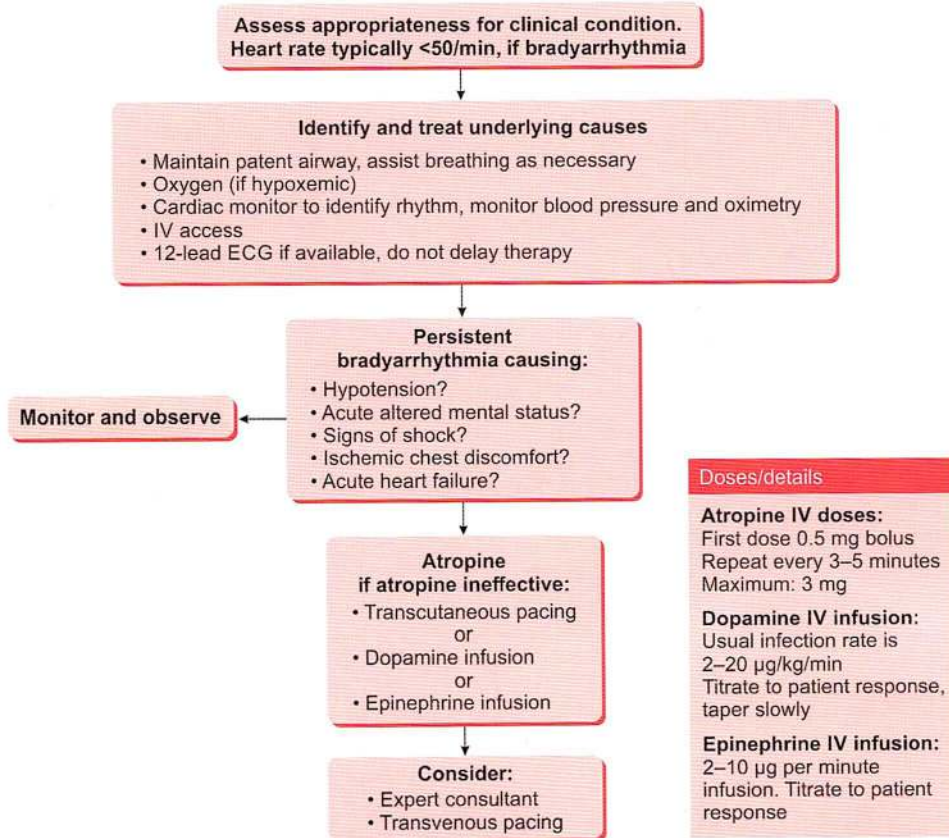
Pathological:

- Raised intracranial pressure (ICP)
- Hypothyroidism
- Obstructive jaundice (action of bile salts on the SA node)
- Glaucoma.

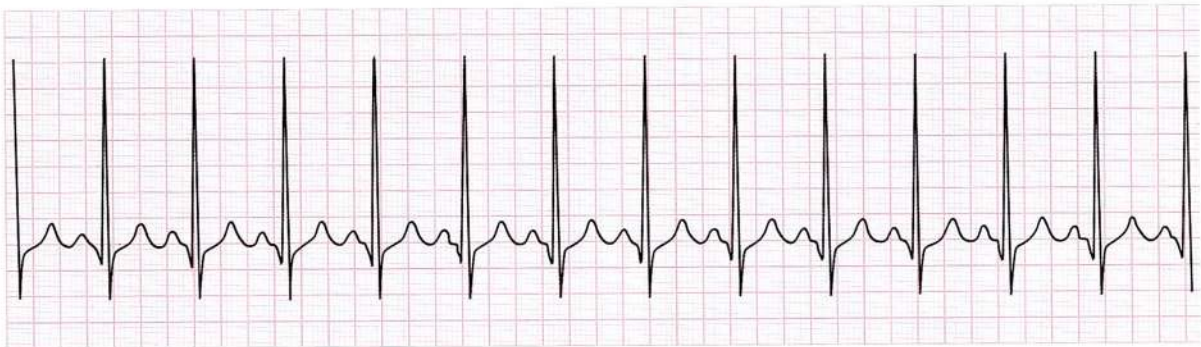
Drugs: Digitalis effect, quinidine, β -blockers, and calcium channel blockers.

Q. What are the Advanced Cardiovascular Life Support (ACLS) 2015 guidelines for management of bradycardia?

Ans. Guidelines adapted from the American Heart Association (AHA)/ACLS guidelines 2015 for the management of bradycardia are shown in **Flowchart 1**.

Flowchart 1: Adult bradycardia with a pulse algorithm.

Source: Adapted from AHA/ACLS guideline 2015.

**Fig. 20:** Sinus tachycardia.**Q. What are the causes of sinus tachycardia?***Ans. Physiological:*

- Exercise
- Pregnancy
- High altitude.

Pathological:

- Pain
- Hypermetabolic states such as thyrotoxicosis and fever
- Anxiety
- Excessive caffeine

- Hemorrhage
- Pulmonary embolism.

Drugs: Atropine and adrenalineExample of sinus tachycardia is shown in **Figure 20**.**Q. What do you understand by paroxysmal supra-ventricular tachycardia? Enumerate the causes and the treatment.***Ans.*

- Circus movement or reciprocating tachycardias utilizing the mechanism of reentry.

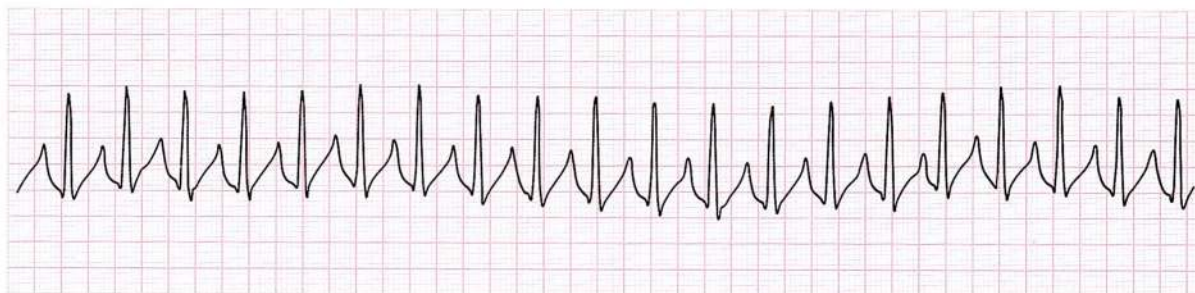
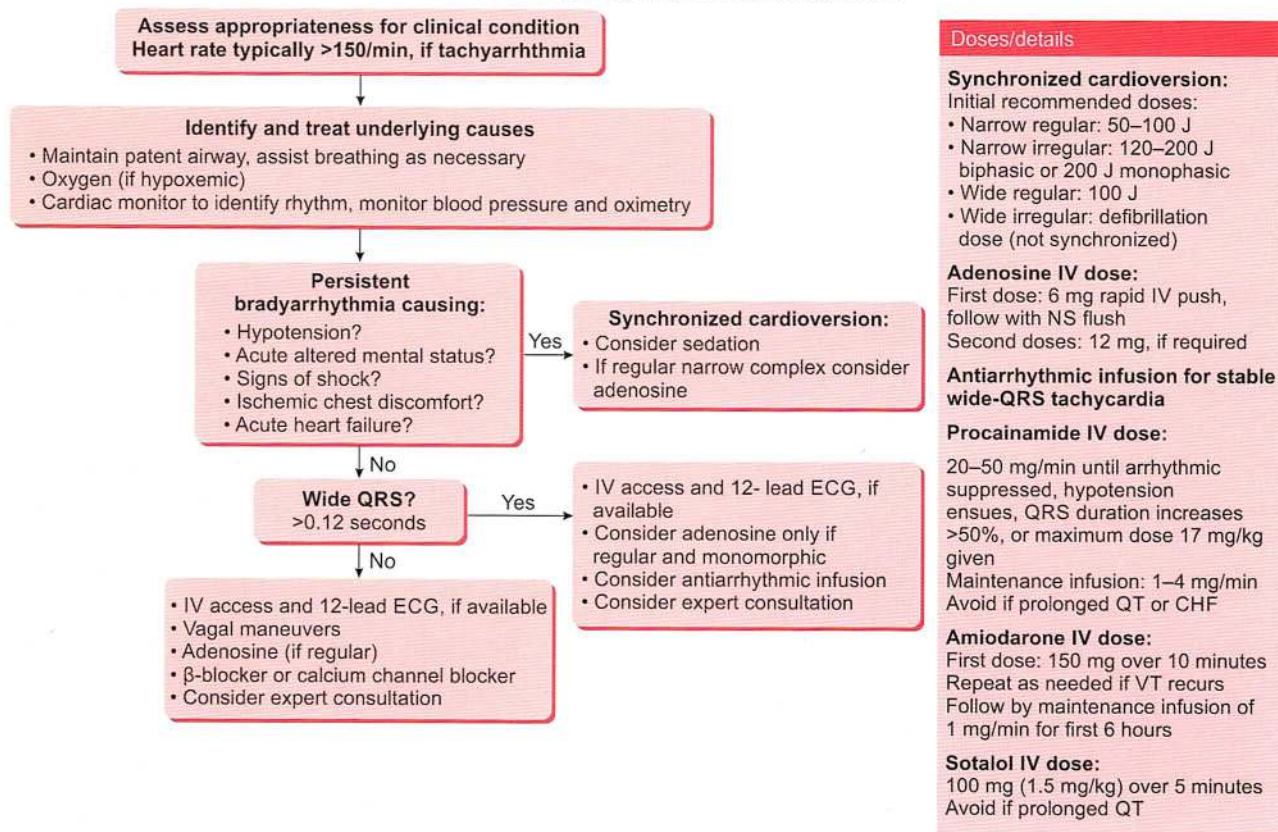


Fig. 21: Paroxysmal supraventricular tachycardia.

Flowchart 2: Adult tachycardia with a pulse algorithm.



(CHF: congestive heart failure; NS: normal saline; VT: ventricular tachycardia).

- The onset is sudden and also stops abruptly—paroxysmal
- Narrow-QRS tachycardia (unless there is preexisting bundle branch block or rate-related aberrant ventricular conduction)
- May be benign
- Persistence would precipitate heart failure.

Causes:

- Idiopathic
- Thyrotoxicosis

- Excessive caffeine intake
- **Drugs:** Adrenaline and amphetamines
- Sepsis.

Example of paroxysmal supraventricular tachycardia is shown in **Figure 21**.

Q. How to manage a patient with tachyarrhythmias?

Ans. Guidelines adapted from AHA/ACLS guideline 2015 for the management of a patient with tachyarrhythmias are shown in **Flowchart 2**.



Fig. 22: Atrial flutter.



Fig. 23: Atrial fibrillation.

Q. Differentiate between atrial flutter and fibrillation.

Ans. Atrial flutter (Fig. 22):

- Rapid and regular excitation contraction of heart
- P wave—saw-tooth appearance
- Can be due to circus movement or focal discharge from an ectopic atrial focus
- Ventricular rate depends on the efficacy of AV conduction.

Atrial fibrillation (Fig. 23):

- Excitation and recovery of atria are disorganized and chaotic
- Causes small twitches of the atrial myocardium
- Irregular ventricular rhythm with absence of P wave
- The atria are functionally fractionated into a chaotic state of islets at various stages of excitation and recovery.

Q. State the causes of atrial fibrillation.

Ans. Causes:

- Rheumatic fever
- Coronary heart disease
- Thyrotoxicosis
- Excessive caffeine.

Drugs: Adrenaline and amphetamines.

Q. What is ventricular tachycardia (VT)?

Ans. Ventricular tachycardia refers to rhythm faster than 150/min where pacemaker is distal to the bundle of His.

Types: Monomorphic (uniform morphology) versus polymorphic versus torsade de pointes.

Monomorphic ventricular tachycardia: A single focus giving rise to the ventricular activation, hence the

electrocardiographic pattern (morphology) remains the same, and the rhythm is called monomorphic ventricular tachycardia (Fig. 24).

Polymorphic ventricular tachycardia: When the ventricular activation sequence varies due to multiple foci, the electrocardiographic pattern differs, and the rhythm is called polymorphic ventricular tachycardia.

Torsade de pointes: A polymorphic VT associated with the long-QT syndromes characterized by phasic variations in the polarity of the QRS complexes around the baseline. Ventricular rate is often >200 bpm and ventricular fibrillation (VF) often results (Fig. 25).

Q. What is ventricular fibrillation?

Ans. Ventricular fibrillation is a disorganized and chaotic activity of the heart resulting in irregular chaotic deflections of various height, shape, and width (Fig. 26).

Q. What are the diagnostic criteria for right atrial enlargement, left atrial enlargement, and biatrial enlargement?

Ans. Right atrial enlargement: Tall and peaked (p pulmonale) P wave amplitude >2.5 mm in lead II and/or >1.5 mm in V₁ (criteria are not specific or sensitive).

Left atrial enlargement: Wide and notched (p-mitrale association with mitral valve disease)—P wave duration >0.12 second in frontal plane (usually lead II).

Biatrial enlargement: Features of both RAE and LAE in the same ECG. P wave in lead II >2.5 mm tall and >0.12 second in duration.

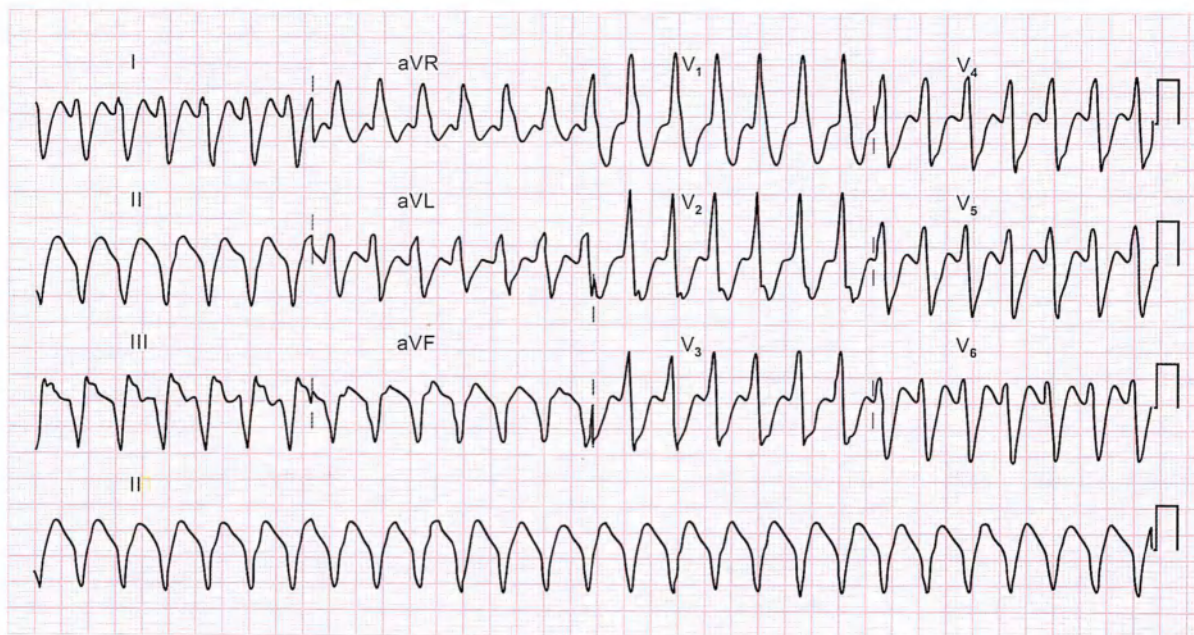


Fig. 24: Monomorphic ventricular tachycardia.



Fig. 25: Torsade de pointes.

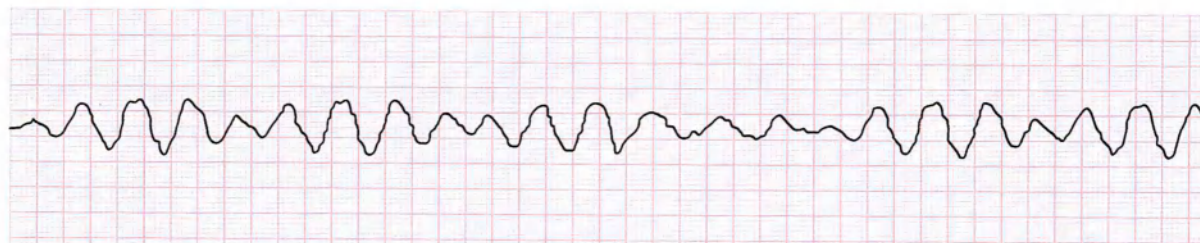


Fig. 26: Ventricular fibrillation.

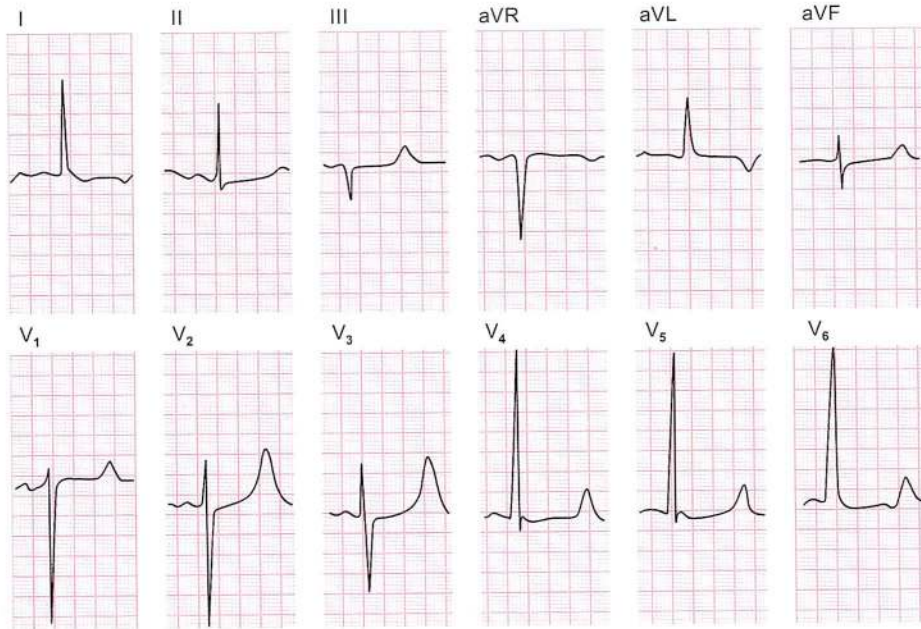


Fig. 27: Left ventricular hypertrophy.

Q. What are the diagnostic criteria and causes for right and left ventricular hypertrophy?

Ans. Causes of RVH:

- Pulmonary hypertension
- Tetralogy of Fallot
- Pulmonary valve stenosis
- Ventricular septal defect (VSD)
- Chronic obstructive pulmonary disease (COPD).

Criteria:

- Right axis deviation
- Tall R wave in right ventricular leads V_1 – V_4
- Right ventricular strain pattern (depressed convex-upward ST segment with an inverted T wave)
- Tall R in aVL.

Causes of LVH (Fig. 27):

- Aortic stenosis
- Aortic regurgitation
- Hypertension
- Cardiomyopathies.

Sokolow-Lyon Index

- S in V_1 or V_2 (leads oriented to RV) + R in V_5 or $V_6 \geq 35$ mm (leads oriented to LV)
- R in aVL ≥ 11 mm (lead oriented to LV).

The Cornell voltage criteria for diagnosis of LVH involves:

- S in V_3 + R in aVL > 24 mm (men)
- S in V_3 + R in aVL > 20 mm (women).

Left ventricular strain: A depressed convex upward ST segment and an inverted T wave in left ventricular leads

V_4 – V_6 . Strain can be due to increased tension within the myocardium or a mismatch between oxygen supply and demand.

Q. What are characteristic features of MI?

Ans. Hyperacute MI: This phase is characterized by ST segment elevation oriented toward the injured site and ST segment depression of the leads oriented toward the uninjured surface. Tall and widened T wave can exceed the R wave.

This phase is quite important as VF occurs most commonly during this phase.

Fully evolved acute myocardial infarction (AMI): Necrosis is reflected by Q waves which are oriented toward the necrotic tissue. Myocardial injury is reflected by the ST segment deviation toward the injured tissue. Myocardial ischemia is reflected by the inversion of T waves oriented toward the ischemic area.

Resolution phase: Gradual return of ST segment to baseline. Tall T waves can appear in leads oriented to uninjured surface. Abnormal Q wave may persist in leads oriented to the injured site.

Anterior wall infarction (extensive anterior infarction): Leads I and aVL, and all precordial leads.

Anteroseptal infarction: Leads V_1 – V_4

Anterolateral infarction: Leads I and aVL, and leads V_4 – V_6 . Example of anterior MI is shown in **Figures 28 and 29**.

Inferior wall MI: Leads II, III, and aVF (**Fig. 30**).

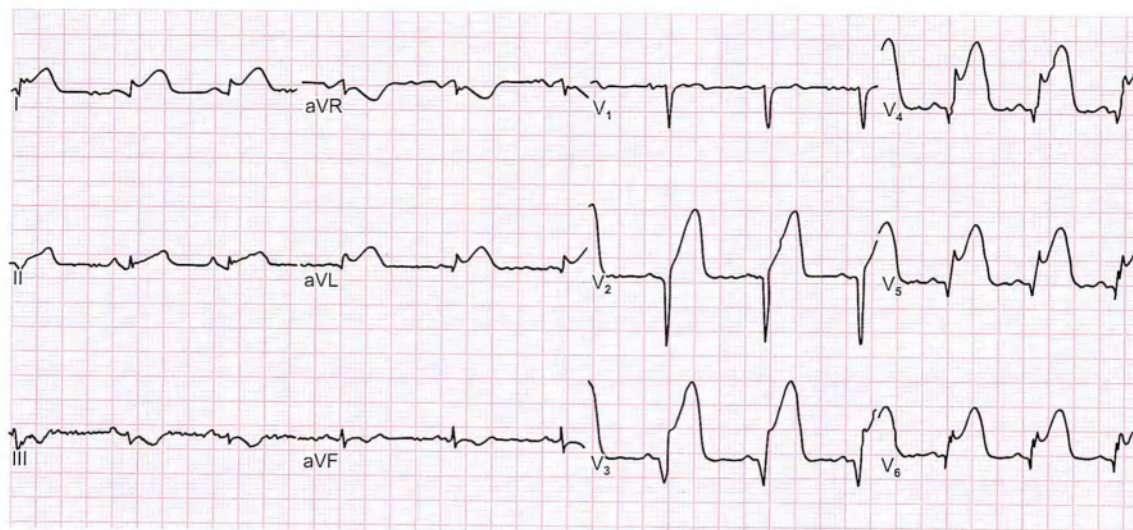


Fig. 28: Anteroseptal myocardial infarction.

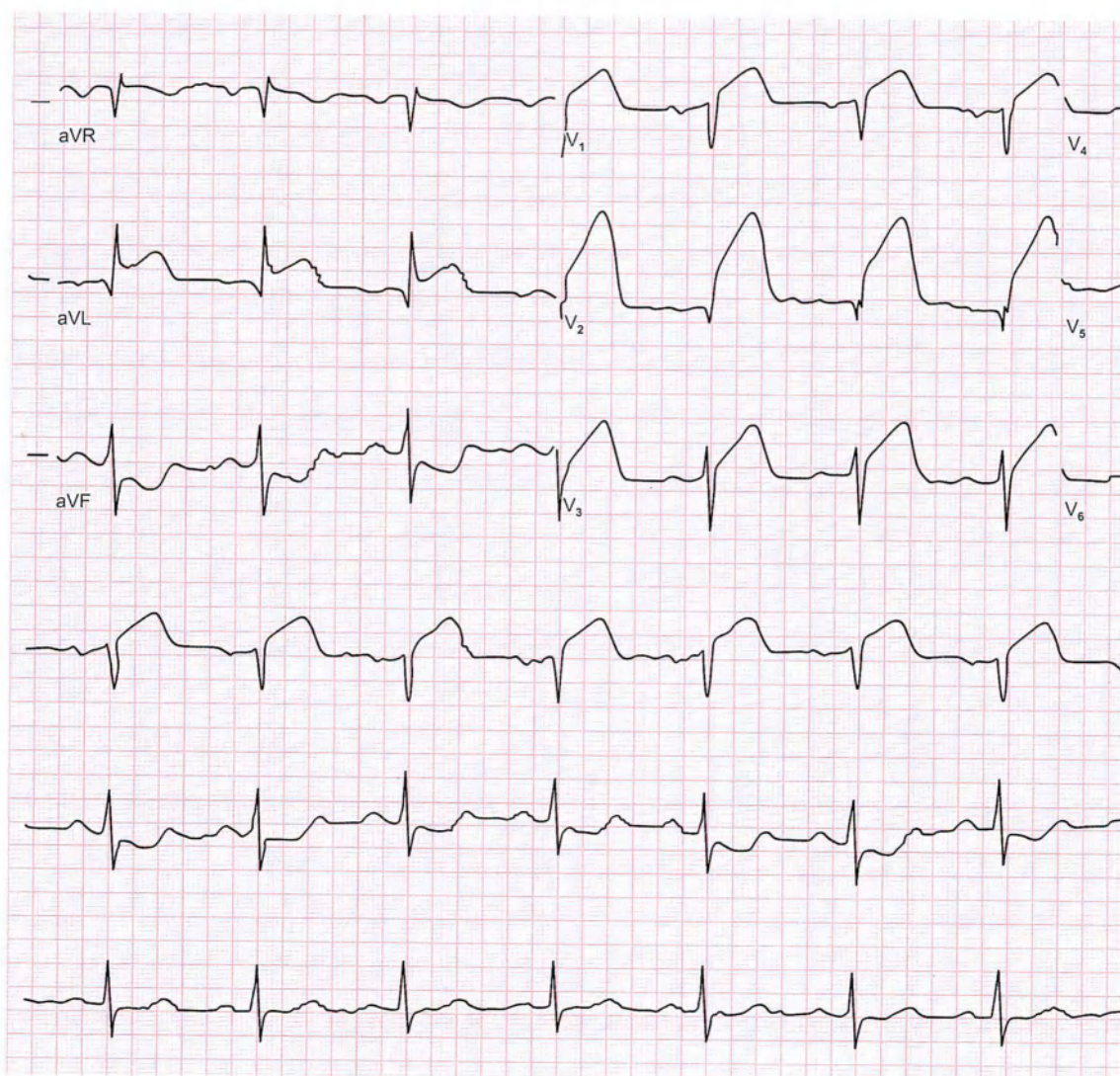


Fig. 29: Anterior wall ST-elevation myocardial infarction.

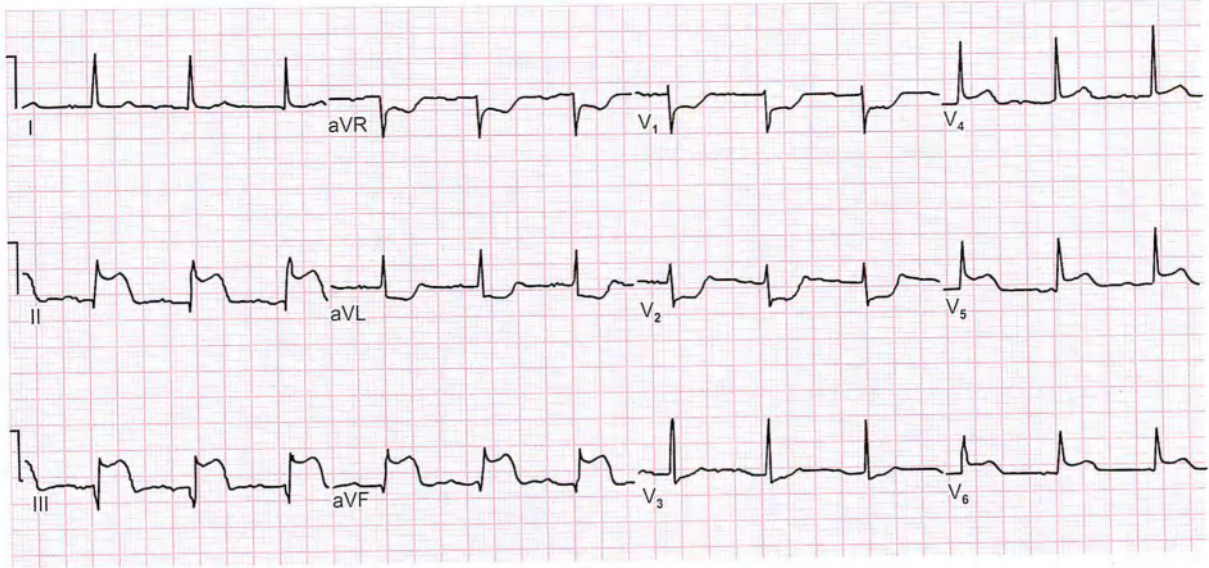


Fig. 30: Inferior wall myocardial infarction.

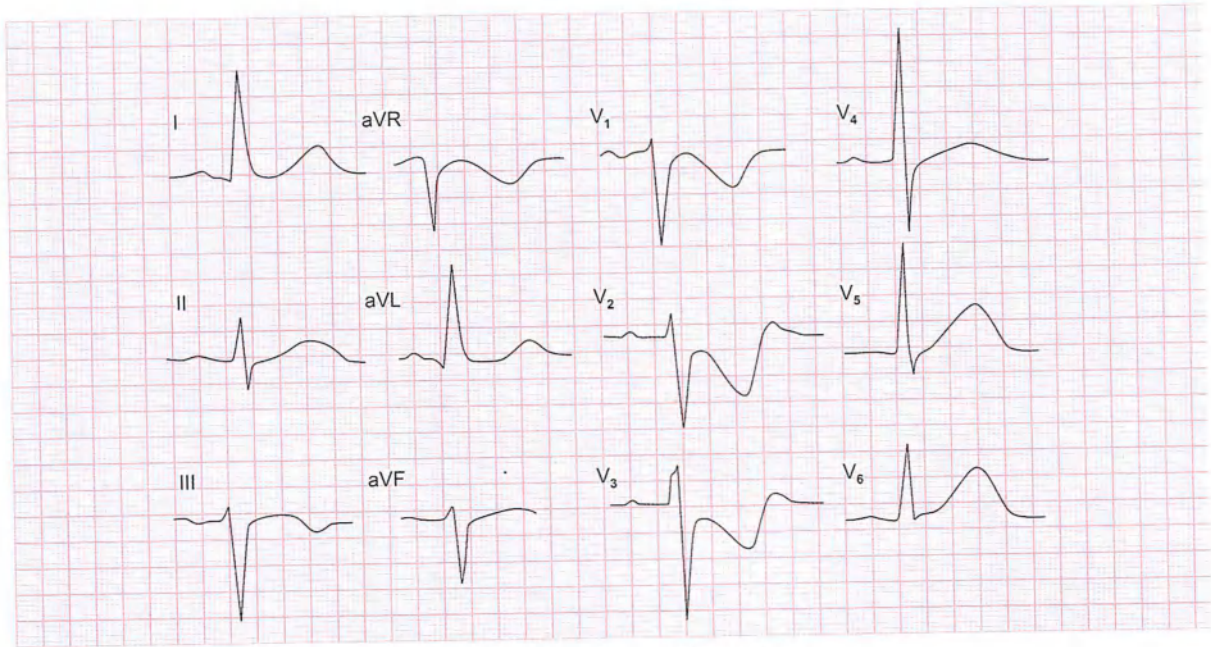


Fig. 31: Posterior wall myocardial infarction.

True posterior infarction: Classical Q wave, ST segment elevation, and inverted T wave do not occur (none of the leads is oriented toward the posterior surface). Diagnosis is made by the reciprocal changes in the leads oriented toward the uninjured surface (anterior surface—V₁ and V₂). Tall and wide R waves, tall T waves, and ST segment depression: V₁–V₂ (Fig. 31).

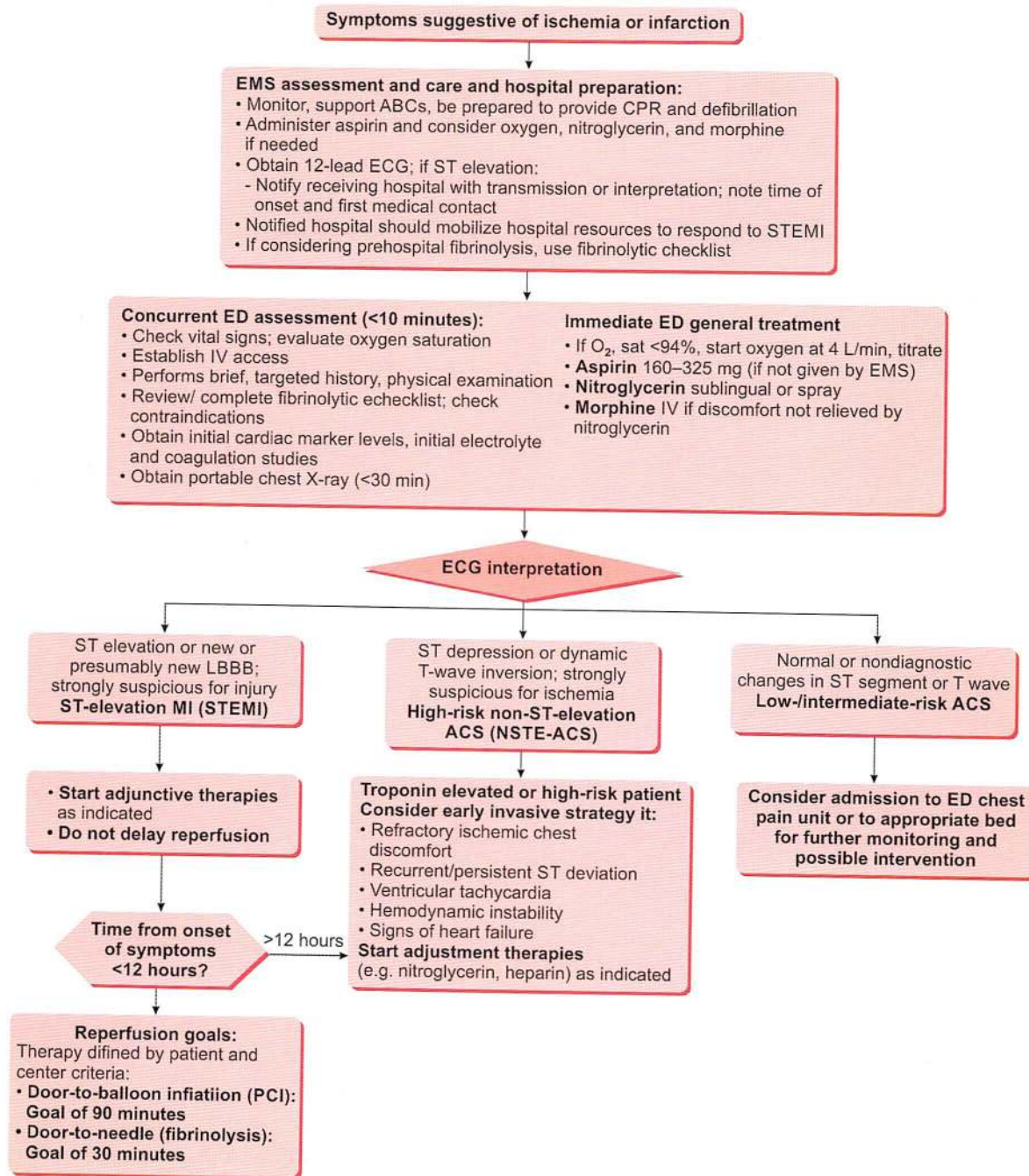
Q. Brief in short the management of acute coronary syndrome.

Ans. The management of acute coronary syndrome is summarized in **Flowchart 3**.

Q. What are the ECG findings in massive pulmonary embolism?

Ans.

- **S1Q3T3 pattern:** Large S waves in lead I, large Q waves in V₃, and T wave inversion in lead III
- Sinus tachycardia is the most common
- Right axis deviation
- T wave inversion in the right precordial leads
- Nonspecific T wave changes and nonspecific ST segment elevation or depression.

Flowchart 3: Treatment of acute coronary syndrome.

(ABC: airway, breathing, and circulation; ACS: acute coronary syndrome; CPR: cardiopulmonary resuscitation; ECG: electrocardiogram; EMS: emergency medical service; IV: intravenous; LBBB: left bundle branch block; PCI: percutaneous coronary intervention; STEMI: ST-elevation myocardial infarction).

Source: Adapted from ACC/AHA guideline 2015.

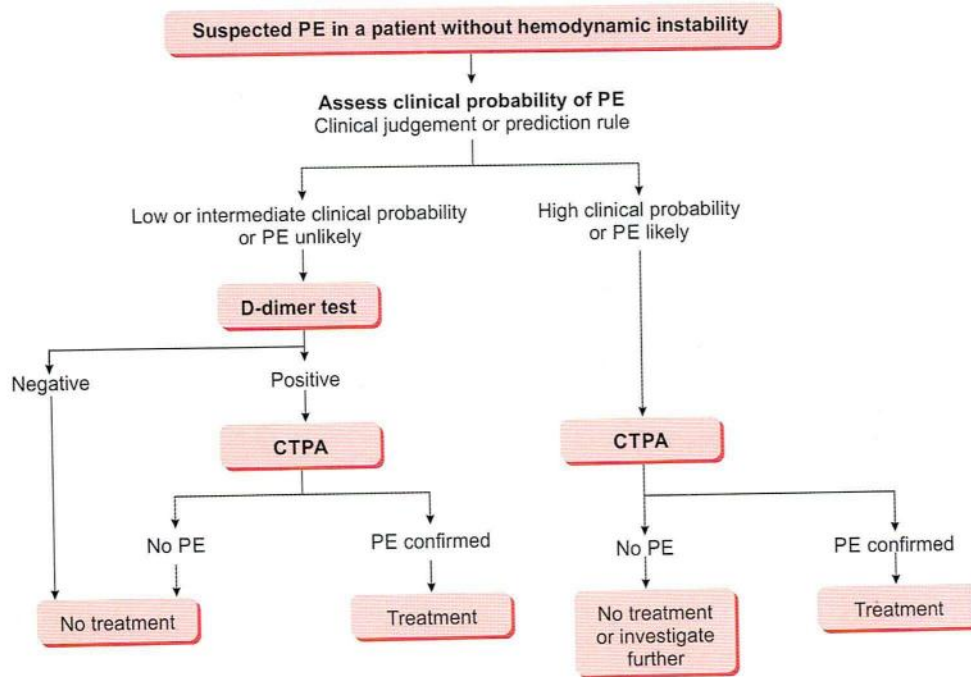
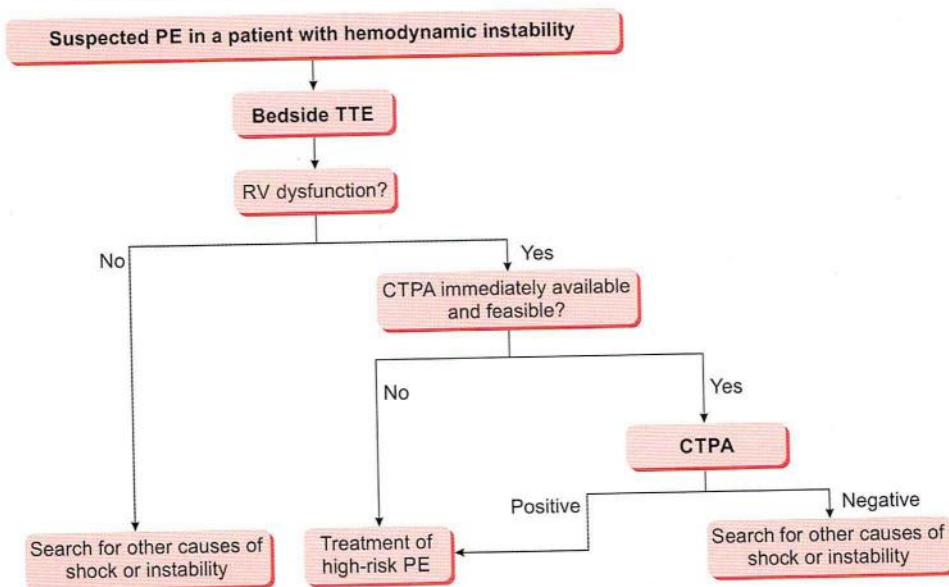
Q. How to evaluate the patient with suspected pulmonary embolism with and without hemodynamic instability?

Ans. Guidelines adapted from 2019 ESC clinical practice guidelines on acute pulmonary embolism are shown in Flowcharts 4 and 5.

Q. Can ECG help in diagnosing pericarditis?

Ans. *Acute pericarditis:*

- Normal voltage
- Raised, concave upward ST segments in leads oriented to heart surface.

Flowchart 4: Suspected pulmonary embolism in a patient without hemodynamic instability.**Flowchart 5:** Suspected pulmonary embolism in a patient with hemodynamic instability.

(CTPA: computed tomography pulmonary angiogram; PE: pulmonary embolism; RV: right ventricular; TTE: transthoracic echocardiogram).
Source: Adapted from 2019 ESC clinical practice guidelines on acute pulmonary embolism.

Chronic pericarditis:

- Generalized low voltage
- Low or inverted T wave in leads oriented to heart surface.

Q. What are the ECG changes associated with digitalis toxicity and quinidine toxicity?

Ans.

- Digitalis toxicity

- Sinus bradycardia
- Premature beats
- *Arrhythmias:* Supraventricular and ventricular
- *Blocks:* First-, second-, and third-degree blocks and bundle branch blocks.

Example: Atrial tachycardia with 2:1 block (Fig. 32).

Quinidine toxicity:

- *Blocks:* First-, second-, and third-degree blocks

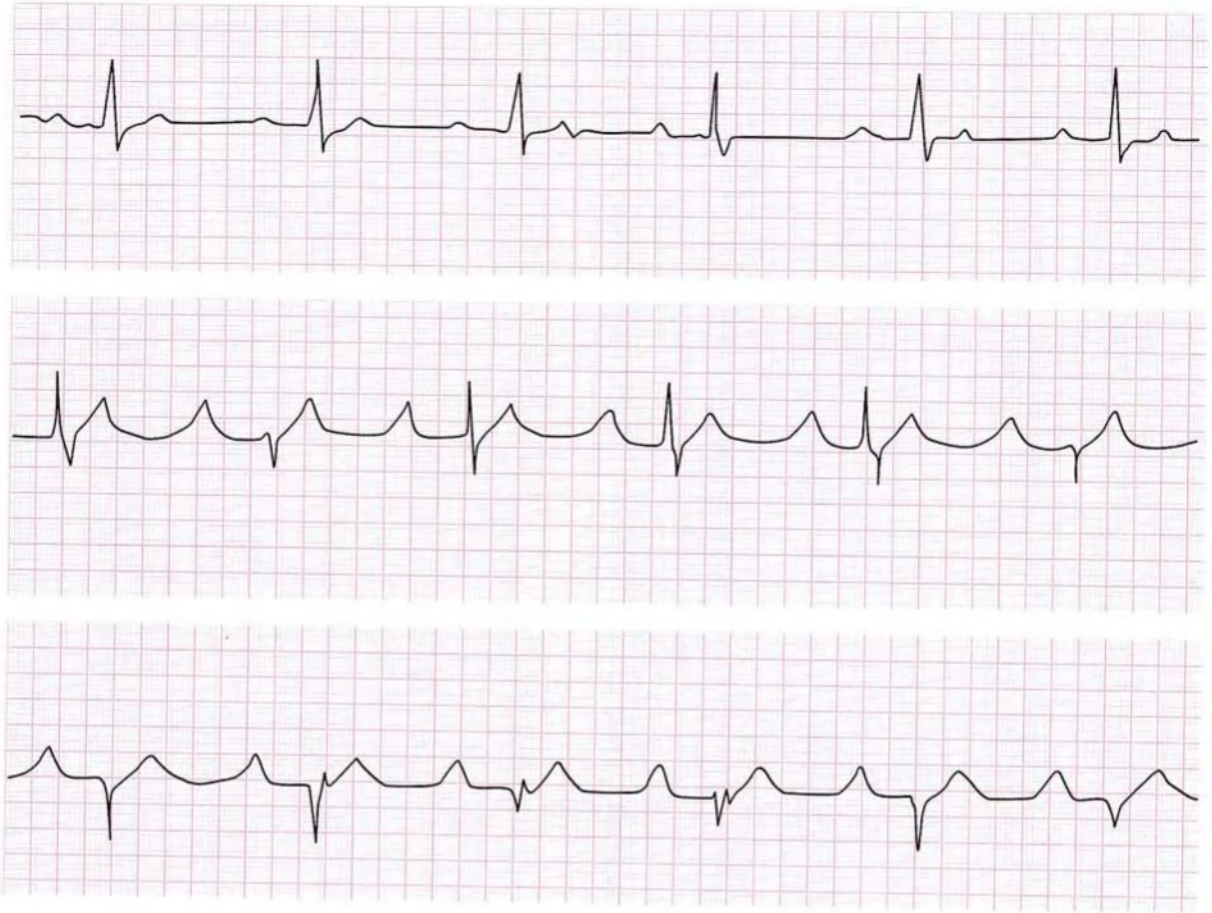


Fig. 32: Atrial tachycardia with 2:1 block.

- Bundle branch blocks
- AV junctional rhythm
- Ventricular premature beats, VT, and VF.

Q. What are the ECG changes associated with hyperkalemia and hypokalemia?

Ans. Progressive changes of hyperkalemia (Fig. 33):

- Appearance of tall, pointed, and narrow T waves
- Amplitude of P wave decreases
- Widened QRS complexes
- ST segment changes (elevation/depression)
- Advanced intraventricular block (very wide QRS with RBBB, LBBB, bi- or trifascicular blocks) and ventricular ectopics
- Absent P waves, very broad and bizarre QRS complexes, AV block, VT, VF, or ventricular asystole.

Electrocardiogram changes of hypokalemia:

- ST segment depression, decreased T wave amplitude, T wave inversion, and increased U wave height
- *Cardiac arrhythmias:* Atrial and ventricular ectopics, atrial tachycardia, and heart blocks
- Increased PR interval.

Hypercalcemia:

- Decreased QT interval and ST segment duration.

Hypocalcemia:

- Increased QT interval and ST segment duration.

Magnesium and ECG:

- Hypomagnesemia resemble that of hypokalemia.
- Hypermagnesemia resemble that of hyperkalemia.

Q. Do changes in temperature affect ECG?

Ans. Electrocardiogram changes associated with hypothermia are as follows (Fig. 34):

- Sinus bradycardia
- Shivering artifact
- Prolonged QT interval
- Prolonged PR interval
- *J wave (Osborne wave):* This occurs when the body temperature falls below 25°C. It appears as an extra deflection at the end of the QRS complex just overlapping the beginning of the ST segment.

Q. What are the ECG changes in hypothyroidism?

Ans. The ECG changes here are due to interstitial myocardial edema and pericardial effusion:

- Sinus bradycardia
- Low-voltage complexes (P, QRS, and T waves)
- ST segment depression

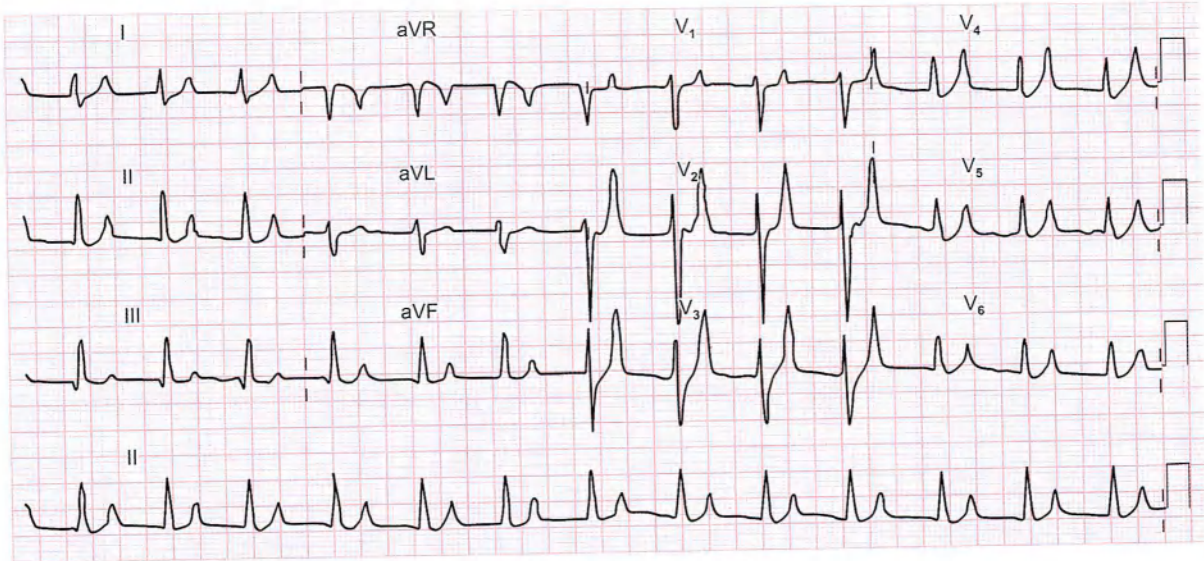


Fig. 33: ECG changes of hyperkalemia.

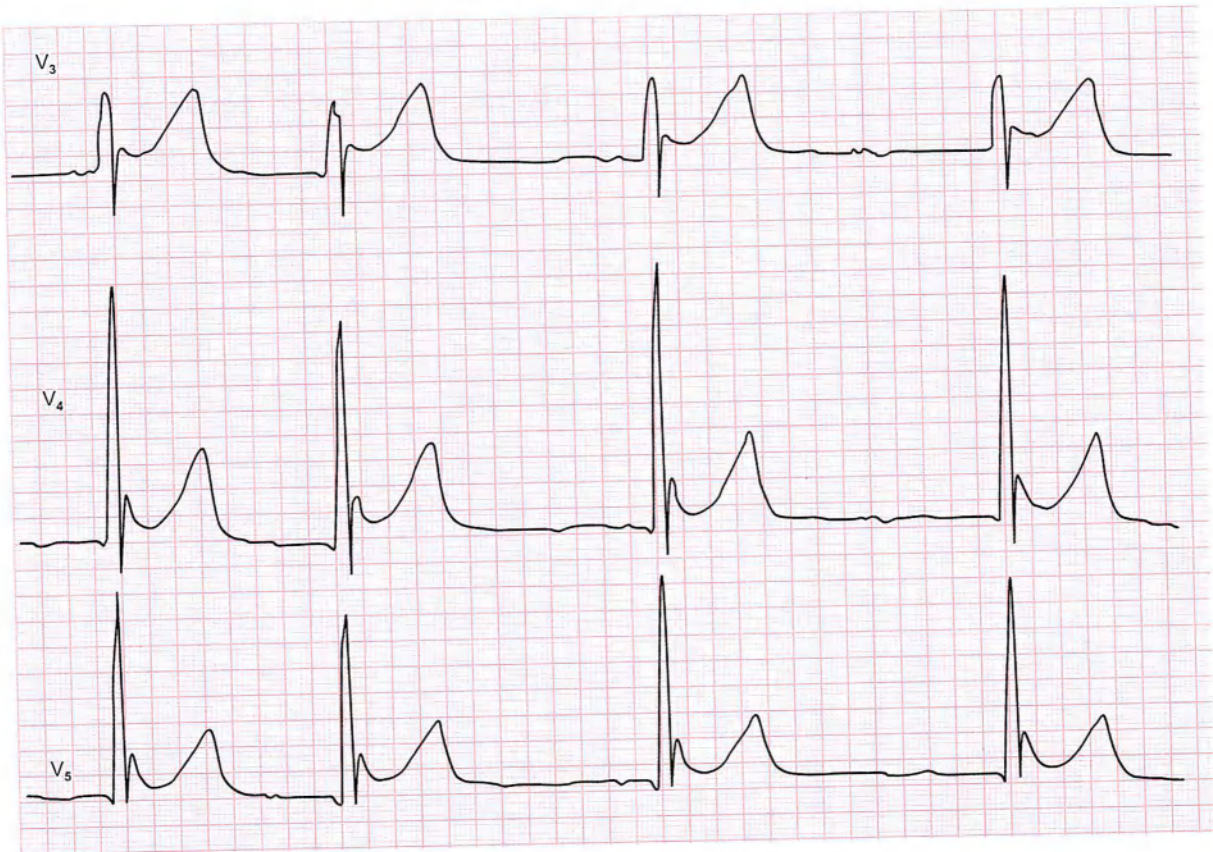


Fig. 34: ECG changes in hypothermia.

- Prolonged PR interval
- AV block
- Prominent U waves
- Corrected QT interval may be prolonged.

Hyperthyroidism

- Sinus tachycardia

- Arrhythmias (atrial fibrillation, atrial flutter, or atrial tachycardia).

SUGGESTED READING

1. ACC/AHA guideline 2015.
2. Schamroth L. An Introduction to Electrocardiography. Hoboken: Blackwell Scientific Publications; 1982.

X-ray Interpretation for the Budding Anesthesiologist

Atul Prabhakar Kulkarni, Aparna Chatterjee, Debashree Lahiri

■ INTRODUCTION

Reading an X-ray is an essential skill for the anesthetist. In the examination, you are likely to be questioned on chest X-rays (CXRs), and X-rays of the cervical spine and abdomen. Sometimes, X-rays of trauma victims are kept and your viva may go in the direction of trauma, diagnosis, and management. It is beyond the scope of this write-up to discuss all the X-rays in full detail. Instead, we will discuss principles of reading the X-rays, and with each X-ray is a list of pertinent questions you may be asked.

■ READING A CHEST X-RAY

X-rays should be read in a systematic manner. Check the following before you read the CXR. The same principles apply to other X-rays.

Label: This is to ensure that you are actually seeing the X-ray of a patient you are interested in. This will generally be in the form of a case record number. What we want is correct patient, correct date and time, and correct (radiology) examination.

Projection: Most CXRs done in the OPD (outpatient department) will be posteroanterior (PA) views (X-ray source behind the patient and the plate placed in front of the chest) taken with the patient standing. Most of those done in the intensive care unit (ICU) (i.e., portable) are anteroposterior (AP) views (X-rays coming from the front and the plate under the patient) with the patient lying down. In AP view, because the mediastinum is close to the X-ray source, it will appear enlarged as the X-rays tend to fan out while reaching the plate. It is therefore difficult to comment on cardiomegaly, unless gross, in an AP view. In the erect view, air-fluid level may be seen in chest (if there is a lesion) or in the gastric fundus. Sometimes, the AP film may be marked as supine. There are several differences in a supine AP view in comparison to a PA erect view, these being smaller lung fields, broader heart and wider mediastinum,

and clavicles projected higher up and horizontal appearing ribs.

Side: The radiographer will always put a side marker on the film. We must check this, if we do not want to miss a dextrocardia or situs inversus.

Then you check the quality of the film by looking at rotation, penetration, timing, and adequacy of the film:

- **Rotation:** To check whether the patient is rotated, see the sternal end (medial end) of the clavicles, they should be equidistant from the midline (spinous processes of the thoracic vertebrae). If the patient is rotated, we cannot comment on the mediastinal shift or compare the quality of lung fields.
- **Exposure or penetration:** In the PA view, you should be able to see the spinous processes of the first four thoracic vertebrae and the intervertebral disc space should be just visible. If the film is overexposed, the lung fields will appear blacker than normal. If underexposed, the lung fields will look too white. Penetration/exposure is important as in an overexposed film, you may miss a pneumothorax.
- **Timing (in relation to respiration):** Most CXRs are end-inspiratory films, you should be able to see 6 anterior ribs or 10 posterior ribs above the diaphragm. End-expiratory films are sometimes done when the radiographer is asked to detect a small pneumothorax. ICU films may be done at any time as the patient may have no control over respiration. In full inspiration, you should be able to count six rib interspaces.
- **Adequacy of film:** You should be able to see the full area of interest. In CXR, you should see both apices as well as both costophrenic angles.

When you describe a CXR, you should say: a labeled CXR with side marked, PA view, well centralized (or not) adequate film with good exposure done in inspiration, before you proceed to actually reading the CXR. Then we can proceed to reading a CXR.

There are numerous ways of going about reading CXRs. The most important thing is to be systematic so that no anatomical structure is missed. We can either proceed from the center (mediastinum) to the periphery or the other way round (**Table 1**). Both these ways are easy to follow.

TABLE 1: Systematic manner of reading a chest X-ray.

Center to periphery	A, B, C, D
Mediastinum	Airway (trachea and bronchi)
Lung fields from apices to diaphragm	Lung fields and bones/soft tissues
Bones and soft-tissues	Heart shadow
Abdominal shadows	D—diaphragm
Foreign bodies	Others

Mediastinum: Check the size, shape, and position of the mediastinum. Check whether the trachea is central. The mediastinum is bound by right brachiocephalic vessels, the ascending aorta, superior vena cava (SVC), and the right atrium and rarely inferior vena cava (IVC) on the right and left brachiocephalic vessel, aortic arch, pulmonary trunk, left atrial appendage, and the left ventricle on the left. The left atrium and the right ventricle are not seen under normal conditions.

Calculate the cardiothoracic ratio: Draw a line in the center (of spine). Draw lines to the right and left heart borders where the distance is maximum from center (commonly designated a and b). Measure the widest thoracic diameter (called c) cardiothoracic ratio from the inner margins of the ribs. The cardiothoracic ratio is given by $(a + b)/c$, normally it is ≤ 0.5 (**Fig. 1**).

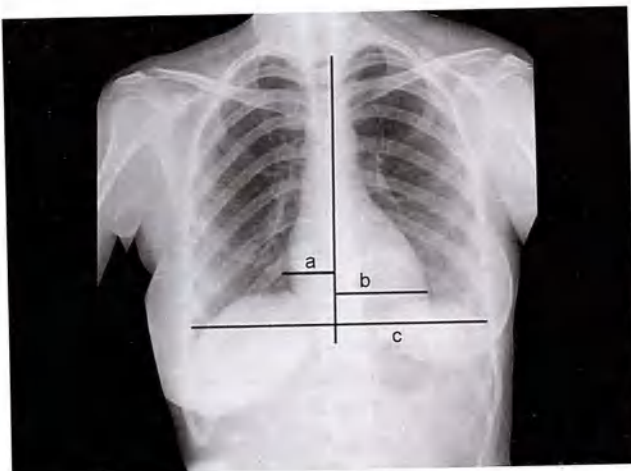


Fig. 1: Cardiothoracic ratio $(a + b)/c$.

Lung fields from apices to diaphragm: Compare the size of lungs and check for proper inflation. Look at the pulmonary

parenchyma, you should find equal radiolucency at similar levels on both sides. Sometimes, it helps if you cover mediastinum with a piece of paper so that it does not distract you from the lungs. The costophrenic and cardiophrenic angles should be clear. For descriptive purposes, the lung fields are nonanatomically divided into upper (above anterior end of the second rib), middle (between anterior ends of second and fourth ribs), and lower (below anterior end of the fourth rib). Look for abnormal radiodensities or radiolucencies, masses, and presence of infiltrates and calcifications. Small lung lesions may be easily overlooked in some areas: the apices, pleural margins, and behind the calcified anterior first rib cartilage, the heart, and the diaphragm.

Check pulmonary vasculature: The left pulmonary artery (left hilum) is higher (1–2 cm) than the right. The vessel size is more in lower than in upper areas (effect of alveolar pressure on low pressure pulmonary vasculature). If this is reversed, then redistribution of blood flow has occurred as in congestive cardiac failure (CCF).

Look at the hemidiaphragms, both should be smooth and curving upward, right slightly higher (2.5 cm) than left. Follow pleura around the rib cage.

Bone and soft-tissues: Check soft tissue shadows, note any obvious mass and calcification, and look for subcutaneous emphysema which may give a hint about underlying pneumothorax and rib fractures. Look at the bony skeleton, note bone densities, and look for masses. Check breast shadows, a missing breast shadow in mastectomy patient may be the only abnormality (normal X-ray for examination).

Abdominal shadows: Check below the diaphragm for bowel gas, organ size, abnormal calcifications, free air, etc.

Foreign bodies: Check for iatrogenic foreign bodies—endotracheal tube (should be above carina), nasogastric tube (below the diaphragm), tracheostomy tube, central venous catheter (CVC) (tip above the level of carina, same as pleuropericardial reflection; if the CVC punctures the SVC wall below this level, cardiac tamponade will occur), intercostal drains (all holes should be inside the chest, none in subcutaneous plane, no kinking, or touching the heart), pacemaker box, lead (check for continuity, note any fractures), and artificial cardiac valves. Then look for other foreign bodies.

■ CHEST X-RAYS FOR PRACTICE

You will find some X-rays below. We will talk about diagnosis/differentials and/or etiologies and then questions pertinent to that diagnosis that you may be asked in the

examination. You should know something of medical theory about each condition and of course all relevant anesthetic/clinical management for that condition. Sometimes in the bracket are the answers.

Chest X-ray 1—foreign body (Fig. 2): This is a very dramatic X-ray with a very obvious diagnosis. You might get a CXR with a needle in one of the bronchi. A systematic review of X-ray will help you to pick it up. The usual questions asked with this X-ray are:

- Where is the foreign body—trachea or esophagus?
- How will you confirm the location (a lateral CXR)?
- How will you anesthetize the patient for removal of (a) tracheal and (b) esophageal foreign body? How will you induce anesthesia (crash induction)?
- Will you use muscle relaxant (preserve spontaneous breathing as far as possible)?
- Differences between organic versus nonorganic foreign bodies, recent versus old (impacted), and central versus peripheral?

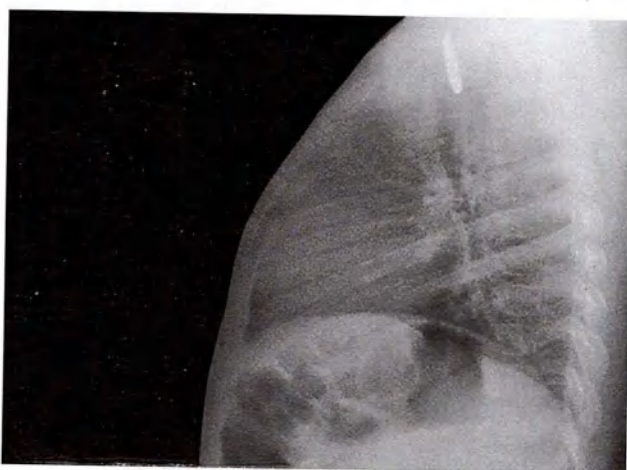
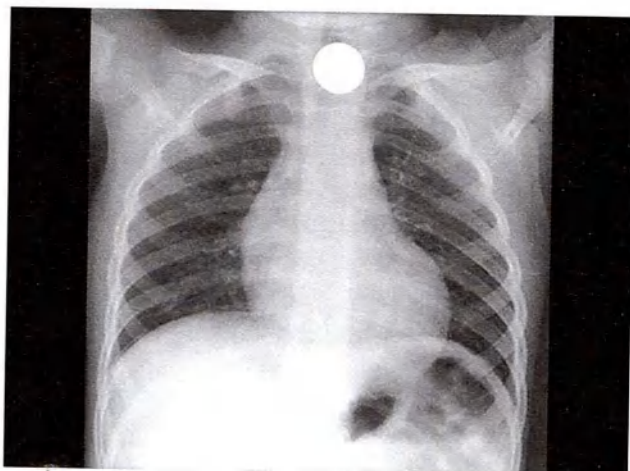


Fig. 2: Frontal and lateral X-rays demonstrate an ingested coin lying in the esophagus.

Chest X-ray 2—pneumothorax (Fig. 3): Chest X-ray (supine position) in a trauma victim showing rib fractures, tension pneumothorax, and subcutaneous emphysema should be done.

The usual questions are:

- What is the etiology of pneumothorax?
- How do you treat a pneumothorax?
- What is tension pneumothorax?
- How do you diagnose it?
- Will you wait for confirmatory X-ray?
- How do you treat a tension pneumothorax?
- How do you differentiate between a tension pneumothorax and cardiac tamponade (clinically)?
- How do you diagnose cardiac tamponade?
- How do you treat cardiac tamponade?
- Describe the technique for insertion of intercostal drain. Describe care of intercostal drain.
- When do you use one bottle, two bottles, etc., for intercostal drainage? Describe care during the transport of a patient with an intercostal drain.

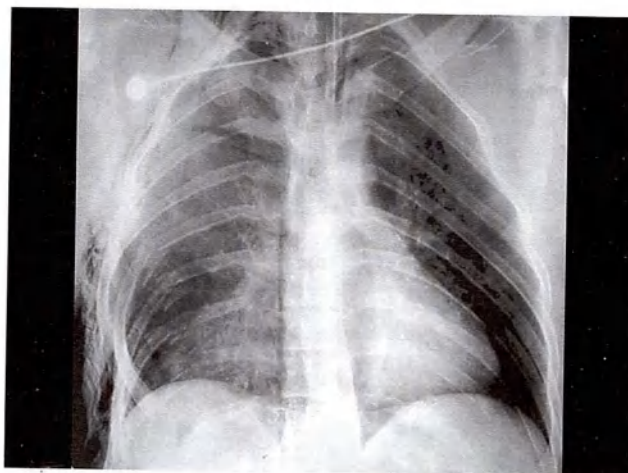


Fig. 3: Pneumothorax.

Chest X-ray 3—pleural effusion (Fig. 4):

- Erect PA view showing left-sided pleural effusion.
- What is the etiology of pleural fluid?
- Why is the top of a pleural effusion C shaped?
- How much fluid should be present in the thoracic cavity for it to be detected on a CXR?
- How do you estimate the amount of pleural effusion?
- Will you drain pleural effusion in all patients?
- Which patients need to have the effusion drained?
- What precautions will you take during induction of anesthesia in a patient with pleural effusion?
- Describe techniques available for drainage of a pleural effusion?
- What is a loculated pleural effusion?
- Why are they difficult to drain?

- How will you differentiate loculated from nonloculated effusion?
- What are the causes of bilateral pleural effusion?
- Do we need to drain bilateral pleural effusions?



Fig. 4: Pleural effusion.

Chest X-ray 4—hydropneumothorax (Fig. 5):

- When the top fluid level in pleural cavity is flat (instead of being C-shaped), it indicates air above the fluid.
- Trauma can frequently lead to hemopneumothorax, so when you see a CXR showing hydropneumothorax, always suspect a trauma case, and look for rib fractures, subcutaneous emphysema, and other additional injuries, if any.
- What are the causes of hydropneumothorax?
- How do you treat hydropneumothorax?
- Does this patient need emergency intubation (if it were a trauma patient)?
- Will you intubate first or put an intercostal drain first?
- Can this be a postpneumectomy X-ray?

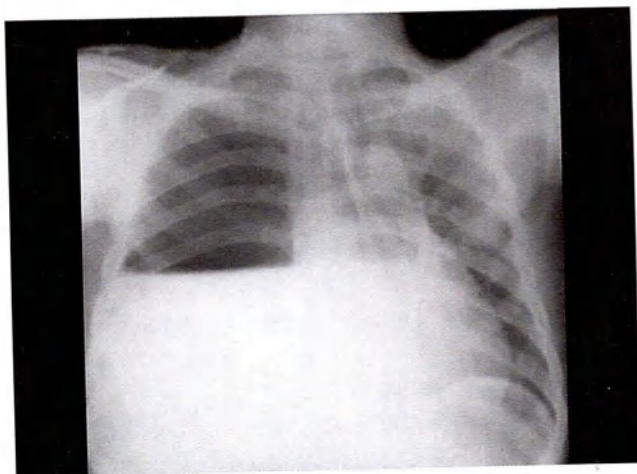


Fig. 5: Hydropneumothorax.

(No, normally the mediastinum shifts to the operated side due to compensatory hyperexpansion of the remaining lung). You may or may not see an intercostal drain.

Chest X-ray 5—opaque hemithorax (Fig. 6):

- What are the differentials of opaque hemithorax?
 - *Lung collapse*: Volume loss and mediastinal shift toward the affected side.
 - *Pleural fluid*: Transudates/exudates, empyema, hemothorax, and chylothorax.
 - *Pneumonectomy*: Look for surgical clips and thoracotomy.
 - *Tumor*: Fibrous tumor of pleura, pleural metastases, and mesothelioma.
 - *Extensive consolidation*: Pneumonia, pulmonary edema, and obstructing neoplasm. Lung agenesis.

The viva will then proceed toward medical management of these conditions and relevant anesthetic management when patient presents with those conditions.

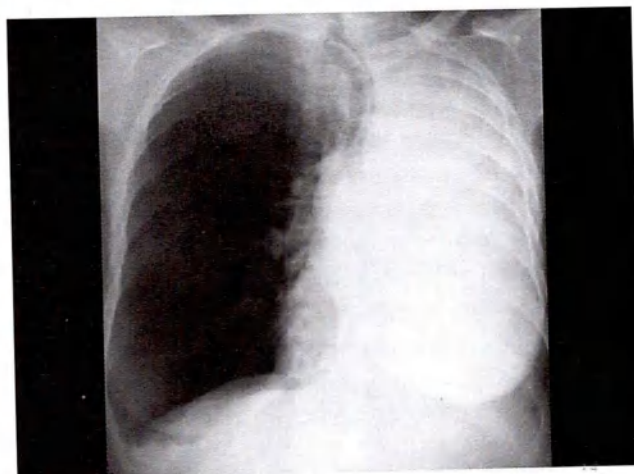


Fig. 6: Opaque hemithorax.

Chest X-ray 6—congenital diaphragmatic hernia (CDH) (Fig. 7):

- X-ray of a child with bowel loops in the hemithorax.
- If you see CXR of an adult with bowel loops in the hemithorax, consider diagnosis of trauma with diaphragmatic rupture hernia and look for other pointers to trauma in the CXR.
- Another differential for bowel loops in the chest is hiatus hernia.
- With CDH, the viva will be for anesthetic management of CDH patients.
- What are the pulmonary problems in CDH patients?
- How do these problems affect anesthesia induction?
- What are the options for management of pulmonary hypertension (PH)?
- Do these patients need postoperative mechanical ventilation?
- How will you ventilate these patients?

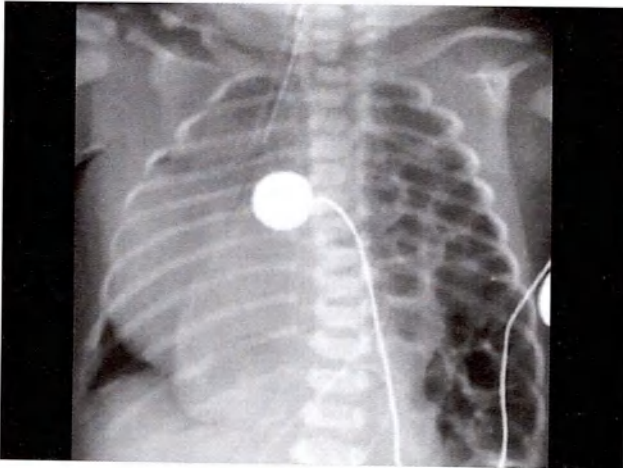


Fig. 7: Congenital diaphragmatic hernia.

Chest X-ray 7—gas under diaphragm (Fig. 8):

- Postoperative patient, so look for evidence of recent surgery (clips, drains, etc.).
- Postlaparoscopy patients, so look for perforated viscus.
- Gas gangrene of the bowel, penetrating abdominal trauma (look for other injuries), and peritoneal dialysis.
- Fallopian tube insufflation and Chilaiditi syndrome (interposition of hepatic flexure of the colon, between the upper border of liver and the right dome of diaphragm may mimic gas under the diaphragm).
- What are the considerations in anesthetic management for emergency laparotomy? What problems do you anticipate? How will you resuscitate these patients? How will you induce anesthesia? Which muscle relaxant will you use?
- What are the likely electrolyte and metabolic disturbances?
- Will you ventilate this patient postoperatively?

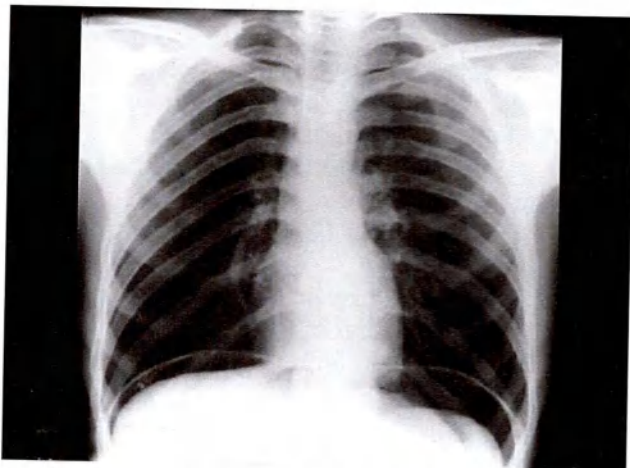


Fig. 8: Gas under diaphragm.

Chest X-ray 8—emphysema with right ventricular hypertrophy (RVH) (Fig. 9): Emphysema is characterized by hyperinflation (>10 posterior ribs above the diaphragm), barrel chest, widened intercostal spaces, depressed or flattened diaphragm, tubular elongated heart, or boot-shaped heart (in case of RVH).

The usual questions are as follows:

- What are the differences in pathophysiology of chronic obstructive pulmonary disease (COPD), asthma, and emphysema?
- How will you investigate this patient for preoperative evaluation?
- What are the spirometric features COPD, asthma, and emphysema?
- What are the bedside pulmonary function tests?
- How do you optimize COPD patients for surgery?
- How will you predict?
 - Likelihood of postoperative pulmonary complications?
 - Need for postoperative ventilation?

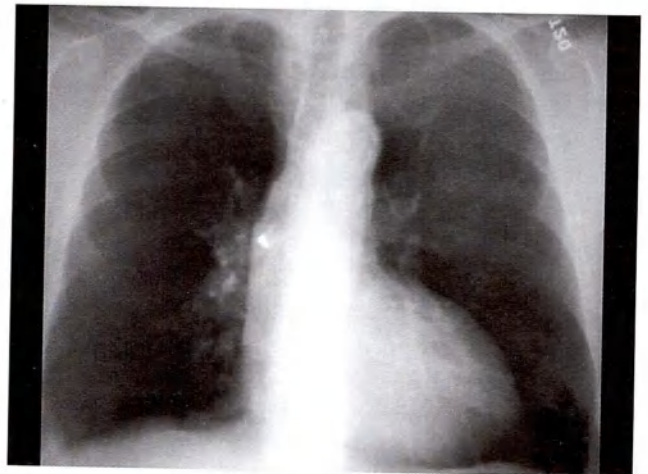


Fig. 9: Emphysema with right ventricular hypertrophy.

Chest X-ray 9—bronchiectasis (Fig. 10): Honeycomb pattern seen at both bases may occur unilaterally. You may get a bronchography X-ray as well showing bronchiectasis. Usual questions are as follows:

- What is bronchiectasis?
- What is the etiology and pathophysiology of bronchiectasis?
- How will you optimize this patient for surgery?
- Discuss various position used for postural drainage in patients with bronchiectasis.
- How will you anesthetize this patient for bronchography?
- What radio contrast media are used for bronchography?
- Describe the bronchography technique.

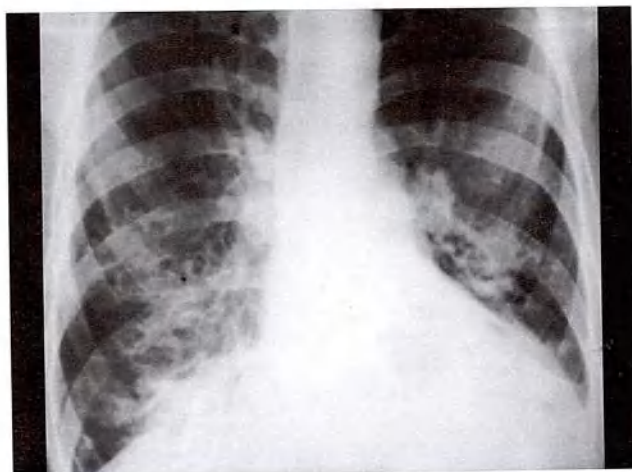


Fig. 10: Bronchiectasis.

Chest X-ray 10—lobar collapse (Fig. 11):

Features: Mediastinal shift toward side of collapse, elevation of the hemidiaphragm, reduced vasculature on that side, hilar mass (if endobronchial tumor), or foreign body (if this has caused collapse), endobronchial intubation could be a cause.

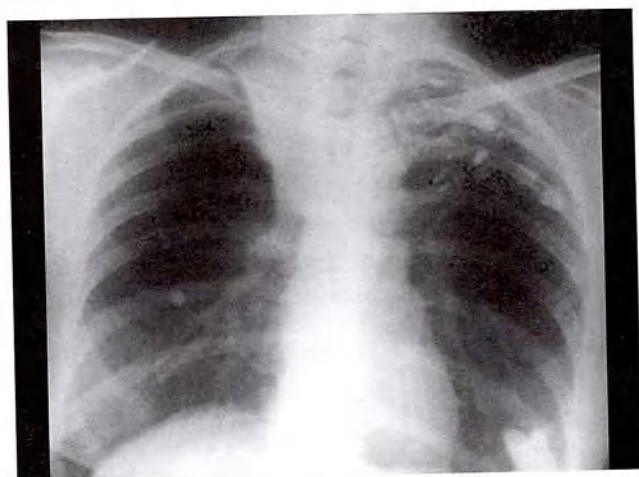


Fig. 11: Right upper lobe collapse.

- What are the causes of lobar collapse?
 - Luminal mass (neoplasm, foreign body, mucus plug/inflammatory exudates, endoluminal metastasis, and misplaced endotracheal tube) bronchial wall inflammation (tuberculosis and sarcoid) and extrinsic compression (lymph nodes and aneurysm).
- What is treatment of postoperative lobar collapse? What is the silhouette sign?
 - An intrathoracic radio-opacity, if it is in anatomic contact with a border of heart or aorta, will obscure that border. If the lesion is not anatomically contiguous with a border or a normal structure, it will

not obliterate that border. Thus, the anterior structure will be silhouetted against the posterior structure.

- What are the causes of postoperative lobar collapse?
- What are the common postoperative pulmonary complications?
- Which patients are susceptible to postoperative pulmonary complications?
- What preventive measures can be undertaken to prevent postoperative pulmonary complications?

Chest X-ray 11—mediastinal mass (Fig. 12):

This will be seen as superior mediastinal widening:

- What is the differential diagnosis of superior mediastinal widening seen on CXR?
- What clinical signs/symptoms will alert you to likely intraoperative problems?
- What findings are you likely to find on spirometry?
- How will you investigate this patient for anesthesia?
- What are the features of fixed/variable and intrathoracic and extrathoracic obstruction on flow-volume loop?
- How does fiberoptic bronchoscopy help in evaluation of these patients?
- How will you induce anesthesia? Intravenous or inhalational?
- How will you intubate this patient?
- What muscle relaxant will you use during induction and maintenance of anesthesia?
- What problems do you expect immediately following induction of anesthesia (airway obstruction/failure to intubate/failure to ventilate)?

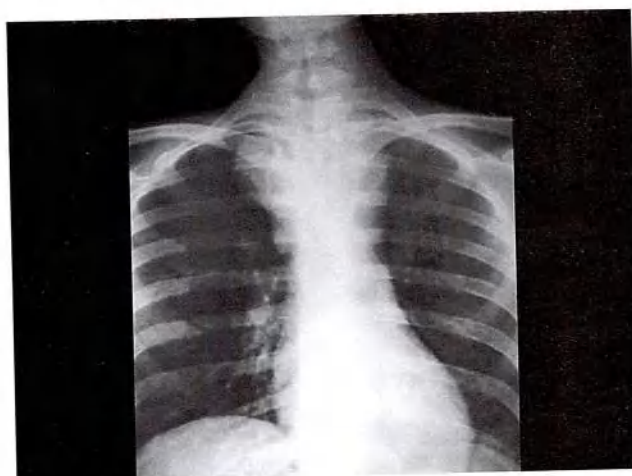


Fig. 12: Mediastinal mass.

- How do you surmount these problems?
- What additional preparation will you keep ready to overcome these problems?
 - Rigid bronchoscope and a competent surgeon, enough people to change patient position to lateral/prone in case of failure to ventilate.

Chest X-ray 12—mitral stenosis (MS) and PH or cardiomegaly (Fig. 13):

- What are the radiological signs of MS with PH?
 - Straightened left heart border, enlarged pulmonary conus, elevation of left main bronchus, “antler” or “mustache sign,” double atrial shadow, indentation of esophagus on barium swallow (lateral X-ray), left lower lobe collapse due to left atrial enlargement, and Kerley B lines.
- What are the types of Kerley lines? (A, B, and C). What does each one signify?
 - Kerley A at least 2 cm unbranching lines coming diagonally from the periphery toward the hila in the inner half, caused by distension of anastomotic channels between peripheral and central lymphatics. Kerley B lines are short (<1 cm) parallel lines at right angles to the pleura, the lung periphery representing interlobular septa. Kerley C lines are short, fine lines throughout the lungs, with a reticular appearance caused by thickening of anastomotic lymphatics or superimposition of many Kerley B lines.
- What are the differentials for cardiomegaly?
 - Ischemic heart disease (IHD), valvular heart disease, congenital heart disease, dilated cardiomyopathy (viral, alcohol, and metabolic), and pericardial effusions.

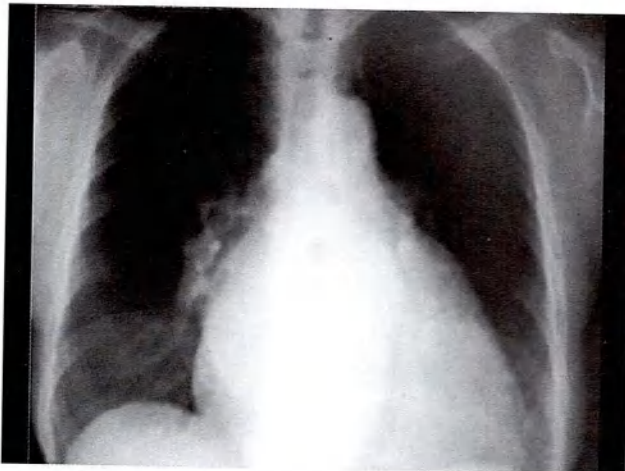


Fig. 13: Mitral stenosis with pulmonary hypertension.

Chest X-ray 13—pacemaker (Fig. 14): This X-ray shows dual lead pacemakers (two). The right one is functioning, obviously placed to replace the fractured (marked with a circle) left-sided leads.

It is most important to trace lead to make sure they are not fractured. Also, look for a pneumothorax (if the X-ray is done postprocedure). The most common site of lead fracture is between the first rib and the clavicle:

- What are the indications for placement of permanent pacemaker?

- How do you diagnose presence of IHD in a patient with a pacemaker?
- What types of pacemakers are available?
- How do you classify pacemakers?
- How do you test that the pacemaker is working properly preoperatively?
- What precautions do you take while anesthetizing the patients with a pacemaker?
- What precautions do you take with the electrocautery? Do you alter pacemaker settings before anesthesia induction? In what way?

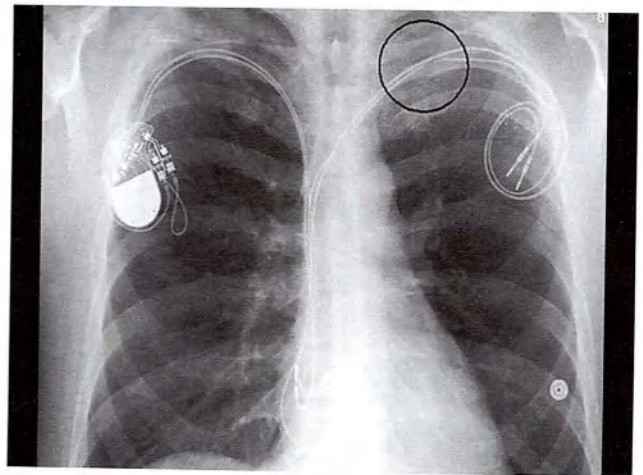


Fig. 14: Pacemaker.

Chest X-ray 14—artificial heart valve (Fig. 15): Presence of sternal wires should alert you to intrathoracic surgery. It is sometimes difficult to distinguish between mitral and

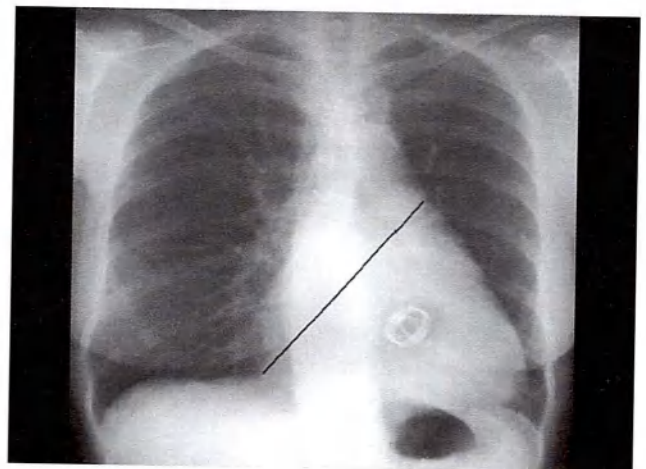


Fig. 15: Prosthetic valve (mitral).

aortic valves. A line drawn from the right cardiophrenic angle to the pulmonary conus helps us do that. A valve below that line is mitral valve and above is the aortic valve.

- How to evaluate patients with prosthetic valves coming for noncardiac surgery?
- How do you manage anticoagulation in these patients? When do you stop warfarin?
- How do you anticoagulate the patient once warfarin is stopped? What are the differences between unfractionated and low-molecular-weight heparin?
- When do you stop heparin before surgery and when do you restart it after surgery?
- What other drugs (antibiotics) do you need to prescribe before surgery?
- How do you manage postoperative bleeding?

the space between first- and second line (retropharyngeal space) beyond 7 mm indicates a retropharyngeal collection, hematoma, or abscess. In such circumstances, patient might have difficulty in breathing and/or difficult intubation. Please also check the height of cervical spines. A compressed fracture may not distort the smoothness of the lines but fracture may be present nonetheless.

Chest X-ray 15—cardiomegaly with pulmonary edema (Fig. 16):

- What are the common causes of cardiomegaly?
- What are the causes of pulmonary edema?
- Cardiogenic vs. noncardiogenic, left ventricular failure (ischemic and cardiomyopathy), valvular heart disease (aortic/mitral), near drowning, aspiration, acute respiratory distress syndrome (ARDS), altitude, raised intracranial pressure, renal failure, and fluid overload.
- How do you treat cardiogenic pulmonary edema? How does positive end-expiratory pressure (PEEP) reduce left ventricular afterload?
- What causes neurogenic pulmonary edema?
- What is negative pressure pulmonary edema? What can cause unilateral pulmonary edema?

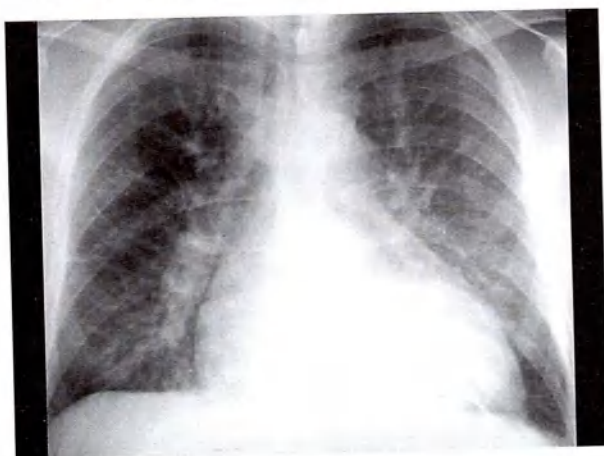


Fig. 16: Cardiomegaly with pulmonary edema.

READING CERVICAL SPINE X-RAYS

Cervical spine clearance is important in trauma patients, particularly those with extensive traumatic brain injury (TBI), and when the spine cannot be cleared clinically. Lateral neck X-ray also gives an idea about the airway. Adequate film of c-spine must include skull base to the upper body of first dorsal vertebra. The smoothness of five lines (marked in the **Figure 17**) indicates preserved integrity of cervical spine (at least nondisplaced fractures). Also, widening of

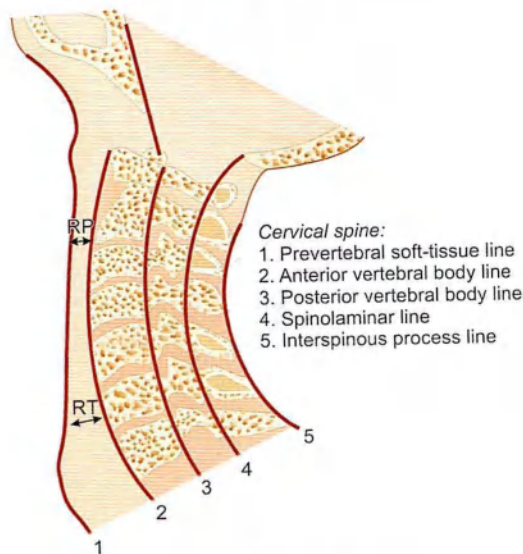


Fig. 17: Cervical spine.



Fig. 18: Cervical spine fracture with widened retropharyngeal space.

Figure 18 shows cervical spine fracture with widened retropharyngeal space. The likely questions with such X-rays will be regarding the management of the airway in a trauma patient, methods adopted for intubating these patients, and other general management of trauma patients.

SUGGESTED READING

1. Corne J, Pointon K. *Chest X-ray Made Easy*. St. Louis: Churchill Livingstone, Elsevier, 2009. Print.
2. Hopkins R, Peden C, Gandhi S. *Radiology for Anesthesia and Intensive Care*. London: Greenwich Medical Media, 2003. Print.

Supraglottic Airway Devices

Vandana Agarwal, Sukhada Savarkar, Atul Prabhakar Kulkarni

Q. Who developed laryngeal mask airway (LMA)?

Ans. Dr Archie Brain developed LMA in 1982 at Royal London Hospital as a modification of the Goldman Dental Mask.

Q. Compare LMA with a facemask.

Ans. In comparison to a facemask the advantages of LMA are:

- It provides a more secure and reliable means of ventilation than the facemask.
- The mandible does not need to be supported; so, hands of the anesthetist are not occupied and do not fatigue.
- It can be easily inserted by a clinical/nonclinical staff as it requires minimal training, particularly during cardiopulmonary resuscitation.
- It can be used in patients with abnormal facial contour provided the mouth opening is adequate for LMA insertion.
- Lower risk of aspiration with LMA (0.012%).

In comparison to a facemask the disadvantages of LMA are:

- Postoperative sore throat (17% vs. 3% for LMA and facemask, respectively).

Q. Compare LMA with an endotracheal tube.

Ans. In comparison to an endotracheal tube (ETT) the advantages of LMA are:

- Reduced stress response as compared to laryngoscopy and intubation and during emergence.
- Minimal increase in intraocular pressure following insertion.
- Reduced requirement of anesthetic agents for airway tolerance.
- Neuromuscular blockers are not needed for insertion.
- It can be easily inserted by a clinical/nonclinical staff as it requires minimal training, particularly during cardiopulmonary resuscitation.
- Lower incidence of postoperative sore throat compared to ETT (45% vs. 17% for endotracheal and LMA, respectively).

- It can be inserted easily in patients with cervical spine injury with immobilization of the head and neck.
- Insertion of LMA does not require laryngoscopy and/or visualization of the vocal cords.
- It provides effective ventilation almost similar to ETT.

In comparison to an ETT the disadvantages of LMA are:

- It does not ensure absolute protection against aspiration but is superior to a facemask.
- It is contraindicated in patients at risk of acid aspiration (e.g., history of gastroesophageal reflux, hiatus hernia, gastroparesis as in diabetes mellitus, inadequate duration of fasting).
- It has a lower seal pressure, so if ventilated with high pressures can lead to gastric insufflation and predispose to regurgitation of gastric contents.

Q. Can you describe a classic LMA?

Ans. It is a supraglottic airway (SGA) device used for anesthesia and maintaining patency of the upper airway. It forms a low-pressure seal around the laryngeal inlet and thus permits gentle positive pressure ventilation.

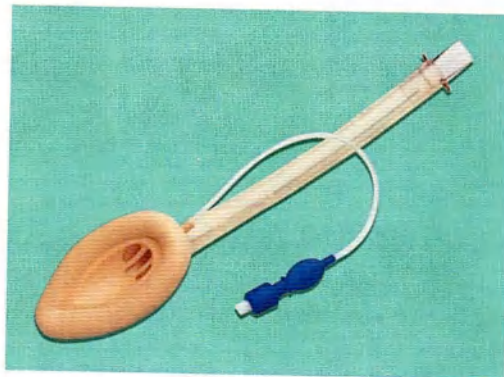


Fig. 1: Classic laryngeal mask airway (LMA).

It consists of an elliptical silicone mask with an inflatable outer rim, a pilot tube and balloon, a latex-free silicone tube, and a standard 15 mm male adaptor attached to the

inflatable outer rim (**Fig. 1**). The elliptical cuff has bars on the laryngeal inlet surface. It can be reused up to 40 times after sterilization by autoclave method.

Q. How will you decide the size of the LMA for a patient and what are the cuff inflation volumes?

Ans. Laryngeal mask airway (LMA) is available in various sizes and depends on the body weight of the patient.

Body weight (kg)	Appropriate LMA size	Air volume (mL)
Up to 5	1	4
5–10	1.5	7
10–20	2	10
20–30	2.5	14
30–50	3	20
50–70	4	30
70–100	5	40
>100	6	50

Q. What preparation is necessary prior to insertion of LMA?

Ans.

- Examine the surface of the LMA for damage, including cuts, tears, or scratches.
- Examine the 15 mm connector to ensure it fits tightly into the airway tube. Do not twist the connector as this may break the seal.
- Carefully insert a syringe into the pilot balloon valve port and fully deflate the cuff so that the cuff walls are tightly flattened against each other. Examine the cuff walls to determine whether they remain tightly flattened.
- Over inflate the cuff with air from complete vacuum and look for any signs of leaks.
- Prior to insertion, deflate the cuff tightly so that it forms a spoon shape. This may be accomplished by pressing the aperture side down onto a flat surface with the nondominant hand. The cuff deflator device can be used.
- Lubricate the posterior surface of the LMA with water-soluble lubricant just before insertion. Lignocaine jelly is not suitable for this purpose as it has been shown to increase the incidence of postoperative sore throat; lignocaine application also anesthetizes the airway predisposing the patient to the risk of acid aspiration postoperatively.

Q. Describe the technique of LMA insertion.

Ans.

- Position the patient with the neck flexed and head extended.
- After the necessary preparation once the patient is anesthetized; grasp the LMA by the tube, holding it like a pen as near as possible to the mask end.

- Place the tip of the LMA against the inner surface of the patient's upper teeth.
- Insert the mask into the oral cavity with the tip directed upward against the hard palate.
- Using the index finger, glide the LMA along the hard palate into the pharynx to ensure the tip remains flattened and avoids the tongue.
- Advance the mask into the pharynx with the dominant hand index finger till it cannot be advanced any further, then with the nondominant hand hold the tube in place and withdraw the dominant hand.
- Gently push the LMA into the pharyngeal cavity to ensure the mask is fully inserted.
- Inflate the mask with the recommended volume of air.
- The LMA rises up slightly out of the hypopharynx; as it is inflated to find its correct position (**Fig. 2**).

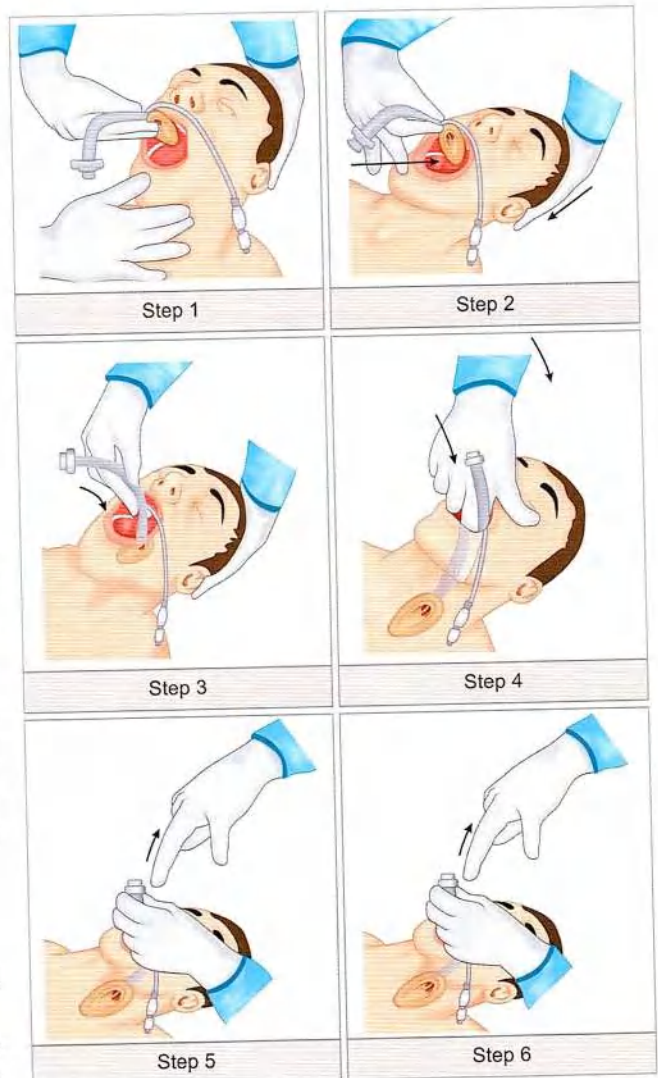


Fig. 2: Technique of laryngeal mask airway (LMA) insertion.

Q. How is the LMA positioned at the laryngeal inlet?

Ans. When appropriately positioned, the distal tip of the silicone cuff rests against the upper esophageal sphincter, the sides of the cuff in the piriform fossa, and the upper part of the cuff against the tongue base.

Q. What are the indications and contraindications for LMA?

Ans. The indications for LMA use are:

- The LMA may be used as an alternative airway during general anesthesia in elective surgical cases.
- It is an essential part of the difficult airway trolley, as it can be used in both anticipated and unanticipated difficult airway to allow ventilation in case there is difficult mask ventilation.
- During cardiopulmonary resuscitation it can be used to secure an airway.
- Relative indication in professional singers; to avoid complications of endotracheal intubation.

The contraindications to LMA use are:

- Any patient at risk of aspiration, e.g., patients undergoing emergency surgery.
- Patients with major local pathology in the pharynx and larynx such as tumor, abscess, edema, and/or hematoma.
- Patient with trismus, facial or upper airway trauma.
- Patients who are morbidly obese, more than 14 weeks pregnant, have received prior opioids medication or any condition associated with delayed gastric emptying.
- Patients with reduced lung compliance/increased resistive work of breathing such as pulmonary fibrosis, status asthmaticus. LMA forms a low-pressure seal around the laryngeal inlet and ventilating patients with these conditions may require higher pressure and so the ventilation may be inadequate. Also, gastric insufflation may occur as ventilation is delivered at higher pressure and gases may leak around the cuff as the sealing pressure is low (20 cm H₂O).

Q. What is the seal pressure for LMA?

Ans. The seal pressure for classic LMA, unique LMA, and flexible LMA is 20 cm H₂O and for ProSeal LMA it is 30 cm H₂O.

Q. What are the common causes of LMA malposition?

Ans. The most common causes of LMA malposition are:

- Placement during inadequate depth of anesthesia and/or suppression of airway reflexes (pharyngeal muscle and/or laryngeal spasm).
- Inappropriate size selection.
- Correct placement may be more difficult in patients with a small mouth, a large tongue and/or tonsils. If larynx is posteriorly placed, it blocks the advancement of the tip of the LMA into the hypopharynx.

Q. What are the complications or adverse effects of LMA?

Ans. The most common complication is postoperative sore throat (the incidence is as mentioned earlier). The other adverse events reported are arytenoid dislocation, trauma to the pharyngeal cavity, airway edema, tongue edema, rarely hypoglossal nerve injury, tongue numbness secondary to lingual nerve injury, tongue macroglossia, recurrent laryngeal nerve injury, inferior alveolar nerve injury, and vocal cord paralysis. The nerve injuries are probably due to malposition of LMA or excessive intracuff pressure, which causes compression of nerves and/or blood vessels. Cuff malposition or excessive cuff pressure may be exacerbated by incorrect mask size, prolonged surgery, and use of nitrous oxide. Simply monitoring the cuff pressure intraoperatively (and evacuating air to maintain constant cuff pressure) was shown to reduce the incidence of postoperative sore throat. A study found that use of a simple device called continuous cuff pressure regulator allowed the cuff pressure to be maintained at a constant level (maximum intracuff pressure < 60 cm H₂O).

Negative pressure pulmonary edema has been reported particularly during emergence from anesthesia. Large subatmospheric negative pressure may develop if the patient tries to breathe against either closed glottis (due to laryngospasm) or against closed tube (when patient bites the tube). Reports also suggest that ProSeal use might be more likely to be associated with this rare but disastrous complication as compared to the other LMAs.

Q. What is the intubating LMA (ILMA or LMA Fastrach)? Can you describe it?

Ans. As the name suggests, it was designed to act as a conduit for endotracheal intubation (**Figs. 3 and 4**). It consists of all

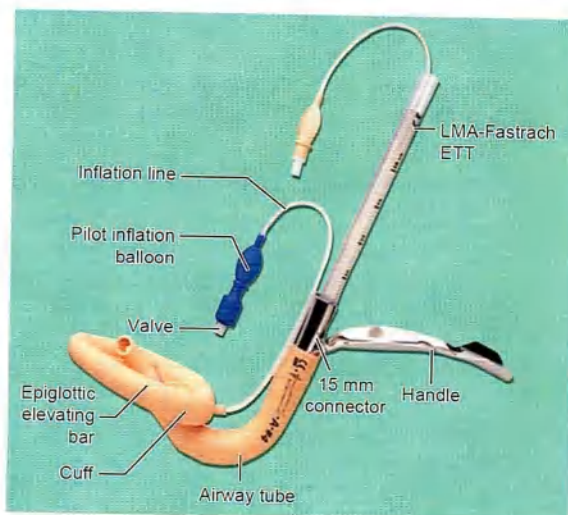


Fig. 3: Laryngeal mask airway (LMA) Fastrach (ETT: endotracheal tube).

the parts similar to the classic LMA; however, there are certain differences. It has an elliptical silicone cuff with rigid anatomically curved steel airway tube cuff which is in turn attached to a rigid handle. The silicone cuff consists of an epiglottic elevating bar instead of the grid as in the classic LMA, which helps in the passage of the ETT. Preparation of the ILMA and patient is similar to the classic LMA.



Fig. 4: Single use laryngeal mask airway (LMA) Fastrach.

Q. What are the available sizes and inflation volume?

Ans.

Body weight (kg)	ILMA size	Air volume (mL)
30–50	3	20
50–70	4	30
70–100	5	40

(ILMA: intubating laryngeal mask airway).

Q. Describe the insertion technique for ILMA.

Ans. After preparation and head in the neutral position, the ILMA is introduced in the oral cavity holding the rigid handle parallel to the patient's chest. Glide the mask along the palate till the straight part of the rigid tube is parallel to the chin, thereafter rotate the rigid handle directing toward the patient's nose till it cannot be advanced any further. Inflate the cuff with appropriate volumes taking care not to exceed the intracuff pressure > 60 cm H_2O . Secure the ILMA in place.

Q. Describe the LMA Fastrach endotracheal tube and the technique of inserting this tube through ILMA.

Ans. Laryngeal mask airway Fastrach endotracheal tube (FETT) is a straight, reinforced (with embedded spiral wire in the wall) cuffed ETT with an internal diameter of 8.0 mm and can be passed easily through the ILMA. The ETT has a marker, a transverse line, at 15 cm; this coincides with the epiglottic elevating bar in the elliptical cuff. It is recommended to use this tube with the ILMA. Using the

conventional ETT through the ILMA can lead to laryngeal trauma.

Prior to insertion, fit the connector to the FETT. Lubricate it well. The LMA FETT has a longitudinal black line on the tube; introduce the tube with this line facing the rigid handle of the ILMA. The FETT should not be introduced beyond the 15 cm mark. Now grip the LMA handle firmly and lift (without levering) it forward by a few millimeters, this increases the seal pressure and optimally aligns the tracheal and FETT axes (the Chandy maneuver). The mask may sometimes have a tendency to flex and the FETT can then pass in the esophagus rather than the trachea, this is avoided by the Chandy's maneuver. Thereafter advance the FETT gently beyond the 15 cm mark; at this point it is lifting the epiglottic elevating bar. Advance the FETT further using clinical judgment. Inflate the FETT and attach it to the breathing circuit to check and confirm endotracheal position.

Q. What is the Chandy's maneuver?

Ans. Chandy's maneuver consists of two steps. The first step is rotating the LMA in coronal and sagittal plane in an attempt to find a position that offers least resistance to ventilation. The second step consists of grasping the handle firmly and use it to draw the LMA forward 2–5 mm in a lifting action without levering on the teeth. This maneuver increases seal pressure and aligns the axes of the trachea and FETT and facilitates blind intubation.

Q. What are the causes of failed intubation through the ILMA?

Ans. The causes are:

- Folding of the epiglottis downward
- Inappropriate size of the ILMA
- Inadequate depth of anesthesia/neuromuscular blockade.

Q. How will you remove the ILMA prior to extubation?

Ans. It is recommended to remove the ILMA following insertion of FETT as it can lead to pharyngeal edema and increased mucosal pressure because of the rigid steel tube in the ILMA. If the ILMA has to be kept in situ, the cuff of the ILMA should be deflated to a pressure of 20–30 cm H_2O .

- With the stabilizer rod measure the distance between the proximal end of the FETT and the patient's incisors.
- Administer 100% O_2 for few minutes and then disconnect the FETT and the breathing circuit.
- Deflate the ILMA completely but keep the FETT cuff inflated.
- Gently displace the ILMA along the pharyngeal curvature till the FETT machine end tip is at the level of the universal connector of the ILMA. Now insert the stabilizing rod in the FETT to keep it in place.

- Remove the ILMA gently over the stabilizing rod until it is clear of the oral cavity.
- Stabilize the FETT to prevent accidental extubation.
- Remove the ILMA and the stabilizing rod and reconnect the FETT to the breathing circuit and confirm position again.

Q. Describe ProSeal LMA.

Ans. The ProSeal LMA (PLMA) (**Fig. 5**) was designed especially for use with positive pressure ventilation at high pressures (in contrast to classic LMA which can be used only with low pressure ventilation). The uses, indications and contraindications, and limitations of ProSeal LMA are similar to the classic LMA. Only the different features of ProSeal LMA are mentioned below.



Fig. 5: ProSeal laryngeal mask airway (LMA).

- The elliptical cuff of the ProSeal extends to the posterior or the pharyngeal side of the PLMA thus improving the seal pressure (dorsal cuff).
- The mask has two tubes attached to it, a drain tube and a reinforced airway tube and a built-in bite block. The double tube arrangement prevents device rotation.
- The mask has a drain tube incorporated within the cuff which communicates with the upper esophageal sphincter permitting drainage of the gastric contents and insertion of gastric tube. The position of the drain tube inside the cuff is designed in such a way to prevent the epiglottis from occluding the airway tube, thus eliminating the need for aperture bars.
- The reinforced airway tube prevents kinking.
- The built-in bite block reduces the possibility of airway obstruction.
- The mask has a strap for PLMA introducer anteriorly between the tube and mask. This also allows insertion of finger or thumb for manual insertion as described for the classic LMA.

- PLMA can also be inserted by inserting a bougie in the drain tube and then passing the bougie in the esophagus after a gentle pharyngoscopy. The PLMA is then guided over the bougie in place. Since the bougie is already in esophagus, the PLMA automatically sits in front of the laryngeal inlet. The bougie may be left in place during surgery, if it is not in the way of the surgeons, this lends stability to the PLMA.

Q. Discuss in brief about LMA supreme.

Ans. LMA Supreme™ (**Fig. 6**) is a latex-free single use LMA similar to the ProSeal LMA. The tube part is rigid, preshaped to adopt to the contour of the anatomy. It also has a fixation tab for securing the LMA after insertion; and a built-in bite block and two ports: (1) the airway port and (2) the drain tube port. The cuff has molded fins which prevent epiglottis from obstructing the airway lumen. The tip of the cuff is reinforced to prevent it from folding during insertion. The patients can be ventilated with pressure up to 37 cm H₂O. Insertion is similar to that of the ProSeal LMA with an introducer, except no introducer is required. The tip has an opening for the gastric tube. Intubation is not possible with LMA Supreme. It is available in both adult and pediatric sizes.

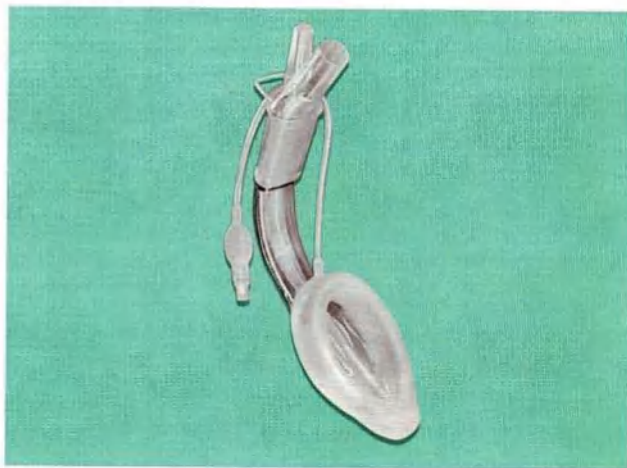


Fig. 6: Laryngeal mask airway (LMA) Supreme.

Q. What other supraglottic airway devices are available? Describe these devices.

Ans. In the past, the choice of an airway device essentially was limited to the facemask or the ETT. Tracheal intubation is the "gold standard" for securing the airway and providing adequate ventilation. However, training in tracheal intubation requires time, appropriate instruments, and adequate circumstances with respect to space and illumination. Furthermore, it requires continued practice and has its own set of complications.

The practice of airway management has become more advanced in recent years. This advancement is

demonstrated by the introduction of new airway devices, several of which have been included in the American Society of Anesthesiologists (ASA) Difficult Airway Algorithm. The standard LMA was introduced into clinical practice in 1988 and rapidly transformed airway management. Since then, there has been an explosion of new SGA devices to compete with the classic LMA, particularly single-use devices. Supraglottic airway is a general term that includes airways with and without sealing characteristics. According to Miller, there are three main sealing mechanisms. The explanation for each provides reasons for the differences observed in the sealing pressures achieved with each of the different types of devices. The indications, contraindications, advantages, and disadvantages of the SGA devices are similar to the LMA. One study found that I-gel produced less increase in intraocular pressure (and LMA Supreme) and lower hemodynamic response than endotracheal intubation.

LMA Classic Excel

It differs from the classic LMA (**Fig. 7**), such as it has an increased angle which aids intubation, armored airway connector with a removal 15 mm adaptor which helps during fiber optic intubation. Because of the reinforce airway tube it can be reused up to 60 times making it more cost-effective. The airway tube can accommodate a maximum ETT size of 7.5 mm ID (through mask size 5 of LMA classic excel). The silicone cuff has an epiglottic elevating bar to aid intubation.

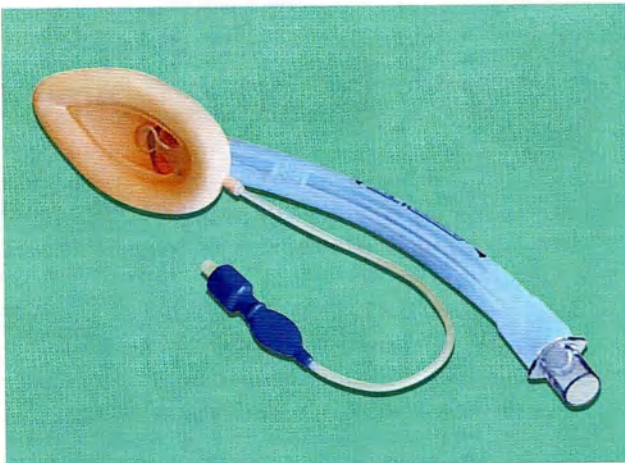


Fig. 7: Laryngeal mask airway (LMA) classic excel.

LMA Unique

It is single use version of classic LMA (**Fig. 8**).

Flexible LMA

As the name suggests, it has a flexible wire reinforced airway tube which makes it suitable for head and neck surgeries (**Figs. 9 and 10**). The length of the airway tube is longer and the diameter smaller compared to the other LMA devices.

The length and the flexibility make it easier to move away from the surgical area. Because of the length it is not suitable for endotracheal intubation. It is available in both pediatric and adult sizes.

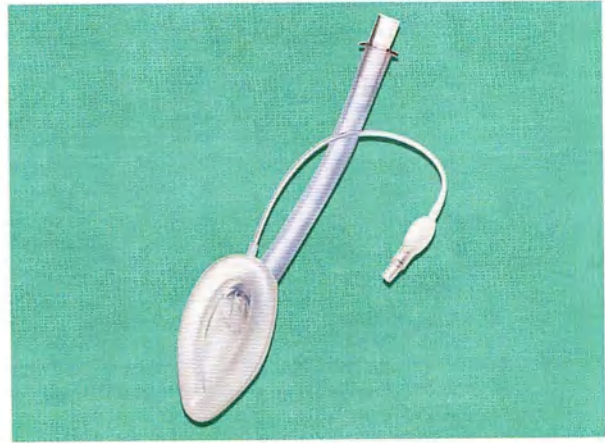


Fig. 8: Laryngeal mask airway (LMA) unique (disposable classic version).



Fig. 9: Flexible laryngeal mask airway (LMA).



Fig. 10: Single use flexible laryngeal mask airway (LMA).

Miller's supraglottic classification:

	Cuffed perilaryngeal sealers		Cuffed pharyngeal sealers		Cuffless preshaped sealers
	Without directional sealing	With directional sealing	Without esophageal sealing cuffs	With esophageal sealing cuffs	
Devices	LMA ILMA SSLM Ambu LM ILA	PLMA	Cobra PLA	LT LTD ETC	SLIPA Baska Mask™
Sealing mechanism	Cuff surrounds the laryngeal inlet. This limits the seal pressure due to direction of forces by the cuff	Forms a seal at the laryngeal inlet and the seal pressure is improved by means of a dorsal cuff which lifts the larynx off the posterior pharyngeal wall	Pharyngeal cuff seals at the base of the tongue		Seals the outlet from pharynx at the base of the tongue right up to the esophageal inlet because of the tough walls Baska Mask the preformed cuff expands with each positive pressure breath

(Cobra PLA: Cobra perilaryngeal airway; ETC: esophageal tracheal combitube; ILA: intubating laryngeal airway; ILMA: intubating LMA; LMA: laryngeal mask airway; LT: laryngeal tube; LTD: laryngeal tube with suction; PLMA: proSeal LMA; SLIPA: streamlined liner of the pharynx airway; SSLM: Softseal laryngeal mask).

Source: Adapted from Miller DM. A proposed classification and scoring system for supraglottic sealing airways: a brief review. *Anesth Analg*. 2004;99:1553-9.

Q. Classify SGA devices.

Ans. The SGA devices can be classified as:

- **Cuffed perilaryngeal sealers:** Ambu laryngeal mask (ALM), Softseal laryngeal mask (SSLM), intubating laryngeal airway (ILA), LMA, and ILMA.
- **Cuffed pharyngeal sealers:** Cobra perilaryngeal airway (Cobra PLA).
- **Cuffless preshaped sealers:** Streamlined liner of the pharynx airway (SLIPA).

Q. Describe the insertion technique of the SGA devices.

Ans. The method of device insertion technique is similar for most of the supraglottic devices and therefore to avoid repetition, a generic method of device insertion is described below. Wherever the technique differs it is mentioned with device.

Insertion Technique

It is important that the individual who inserts and/or ventilates through SGA is familiar with the warnings, precautions, indications, and contraindications in the use of that particular device. Before insertion, the following points are of the highest importance:

- The size of the SGA must be appropriate for the patient. Use the SGA recommendations and clinical judgment to select the correct size.
- If the device is too small, clinicians tend to overinflate the cuff to achieve seal, this may lead to complications.
- Check the SGA cuff and lubricate with water soluble lubricant as directed.
- Do not use excess force for insertion.

- Preoxygenate and implement standard monitoring procedures.
- Ensure adequate depth of anesthesia before attempting insertion. Trainees or inexperienced users should insert the SGA when the patient is at a deeper plane of anesthesia.
- If not contraindicated, use the "sniffing position" (neck flexed and head extended) for insertion.
- The device should be held between the fingers and thumb of the dominant hand, the line on the tube aligned midline and direct it against the hard palate. One may have to use jaw lift maneuver for insertion. The airway should be advanced into the oropharynx with gentle twisting motion to negotiate the tongue and tonsils. With the nondominant hand hold the 15 mm connector and push the airway caudally until resistance is encountered.
- The cuff should be inflated with air until a "just-seal" pressure is obtained. To ensure an adequate seal and minimal trauma it is recommended to maintain intracuff pressure to a maximum of 60 cm H₂O. Pressure higher than this increase the incidence of sore throat and may cause nerve injuries as well.
- After the cuff inflation the airway or tube moves out slightly and fullness is noticed in the neck indicating correct placement.
- The airway or tube is connected to the breathing system and adequacy of ventilation assessed.
- **Indicators of correct SGA placement are:** Auscultation of bilateral lung sounds, bilateral chest excursion, absence of gastric insufflation, and capnography.

- A bite block may be inserted to prevent tube occlusion during emergence from anesthesia.
- Tube should be taped on to the patient's face.

Q. Describe the Ambu laryngeal mask.

Ans. It is a cuffed perilaryngeal sealer. The ALM (**Fig. 11**) is a sterile, single use product made of polyvinyl chloride (PVC), molded in one piece. It has a built-in curve replicating the anatomy of the oropharynx-hypopharynx. The curve ensures that the patient's head remains in a natural, supine position when the mask is in use. It has a reinforced tip, which along with the curve, facilitates insertion of the mask. The internal ribs in the curve make the airway tube flexible to adapt to individual anatomical variations and head positions. The cuff is elliptical and when inserted rests in the hypopharynx at the base of the tongue. The cuff does not have epiglottic bars and this allows easy access for fiberoptic bronchoscope. It has a single inflation line. It is latex free and is available in both adult and pediatric sizes. The proper size is based according to the weight and size of the patient. Ambu® Aura40™ is a reusable Ambu LMA, whereas Aura-i™ and Aura Once™ are disposable products. Ambu Aura Flex™ has a flexible tube without a built-in curve.

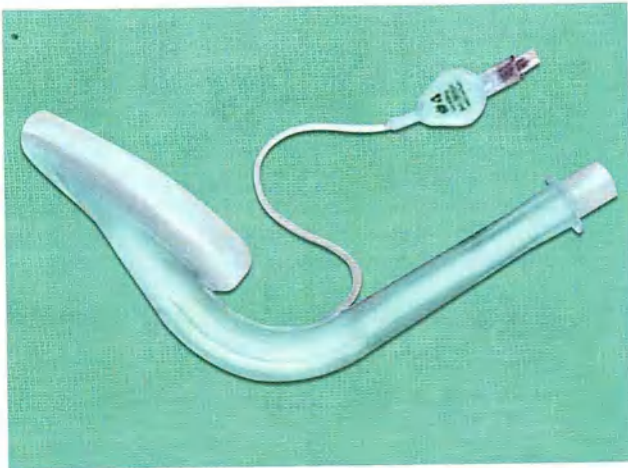


Fig. 11: Ambu laryngeal mask.

Different sizes of Ambu laryngeal mask according to patient weight:

Patient weight (kg)	Size	Maximum cuff volume (mL)
30–50	3	20
50–70	4	30
70	5	40

Q. Describe Ambu® AuraGain™

Ans. Ambu AuraGain (**Fig. 12**) is a single use, second-generation supraglottic device made up of PVC. It is anatomically shaped and has a soft rounded curve, which

allows easy placement. The cuff of the device is thin and made up of soft PVC material which conforms to the airway of the patient and can deliver high seal pressures. Its built-in bite absorption area helps in preventing airway occlusion. It has an integrated gastric channel with a low friction inner surface that allows rapid placement of a wide bore gastric tube. Both adult and pediatric sizes are available based on the weight of the patients. Ambu AuraGain can also act as a conduit for endotracheal intubation. The airway tube of Ambu AuraGain is wider and can accommodate a maximum ETT size of 8.0 mm ID (through mask size of 5 and 6). The airway tube also has integrated navigation marks which provide a guidance for inserting flexible endoscope. Ambu AuraGain is made up of latex-free and phthalate-free material and it is MRI safe.

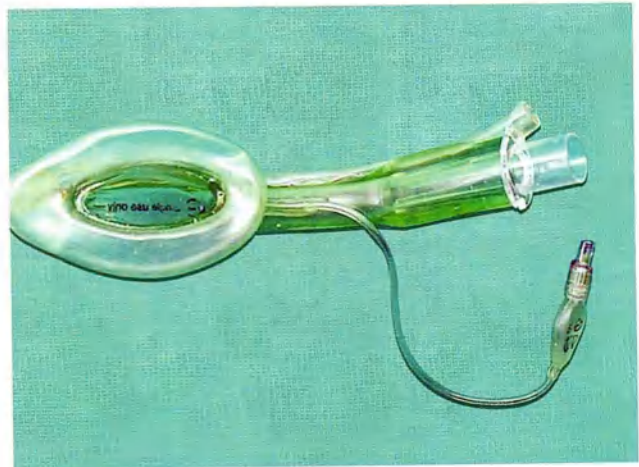


Fig. 12: Ambu AuraGain.

Size selection, maximum cuff volume, maximum ET tube, and maximum gastric tube size which can be passed through different sizes of Ambu AuraGain:

Size	Patient weight	Maximum cuff volume (mL)	Maximum ET size	Maximum gastric tube (Fr)
1	<5 kg	4	3.5	6
1.5	5–10 kg	7	4.0	8
2	10–20 kg	10	5.0	10
2.5	20–30 kg	14	5.5	10
3	30–50 kg	20	6.5	16
4	50–70 kg	30	7.5	16
5	70–100 kg	40	8.0	16
6	>100 kg	50	8.0	16

Q. Describe the cuffed pharyngeal sealers—laryngeal tube and the Laryngeal Tube Suction™ (LTS).

Ans. The LT consists of a silicon reusable airway tube provided with two cuffs (pharyngeal and esophageal) a single balloon for pressure control and a 15 mm standard adapter, which is designed to be reused up to 50 times.

The airway tube is short and “J” shaped, has an average diameter of 11.5 mm and a blind tip (**Fig. 13**). It is suitable for neonates to large adults. LT size selection is based on the patient’s weight for infants and children and height for adults.

Size	Use	Color
0	Newborn < 5 kg	Transparent
1	Infants 5–12 kg	White
2	Children 12–25 kg	Green
3	Adults with height <155 cm	Yellow
4	Adults with height 155–180 cm	Red
5	Adults with height >180 cm	Violet

After device insertion, the proximal cuff lies in the hypopharynx and the distal in the upper esophagus. Both cuffs are high volume and low pressure to avoid ischemic damages and permit a good seal. The proximal cuff is protected by a bend located in a “V” dent of the pharyngeal cuff. With cuff inflation, soft tissue is deflected from this opening, helping to maintain a patent airway. A wedge-shaped block closes the tip of the tube, thus diverting the ventilation to the trachea. The cuffs should be inflated, with the aid of the laryngeal cuff pressure gauge, to 60 cm H₂O. Due to single inflation line, both cuffs will inflate simultaneously.

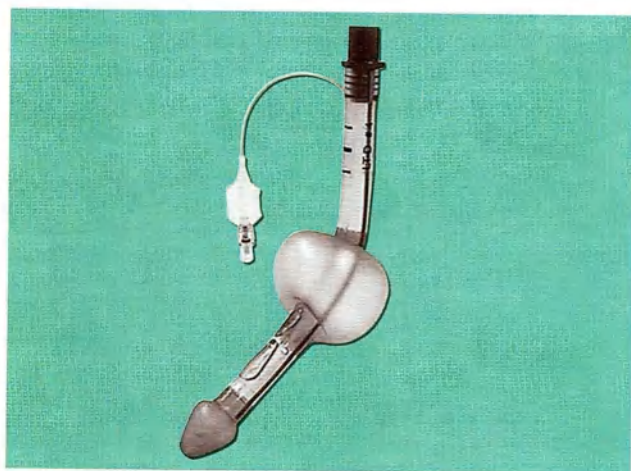


Fig. 13: Laryngeal tube.

Three side eyelets on each side allow improved collateral ventilation; should the epiglottis obstruct the main ventilation orifice. This version has also been manufactured 1.0 cm longer to facilitate deeper placement of the device, thus assuring proper positioning of the two main ventilation orifices and location of the proximal cuff beneath the tongue so that the epiglottis is more likely to be raised with cuff inflation in much the same manner as with a Macintosh laryngoscope blade.

The LTSII™ (**Fig. 14**) is double lumen silicon version of the LT. The additional lumen is situated behind the lumen for ventilation. This lumen is for insertion of gastric tube allowing suction of gastric contents.

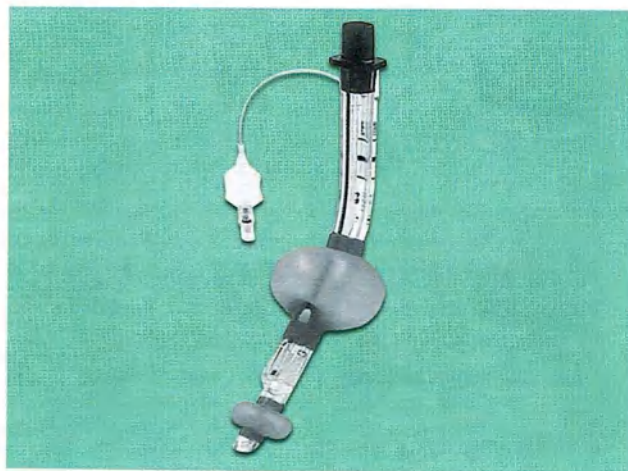


Fig. 14: Laryngeal tube suction.

Insertion Technique and Device Removal

- The device has to be steam autoclaved, disposable versions are available.
- The LT should be held in the dominant hand and inserted blindly along the midline of the tongue sliding the tip along the hard palate until resistance is felt.
- The cuffs can then be inflated with the cuff pressure gauge up to 60 cm H₂O. Alternately a syringe can be used, cuff inflation volumes vary with the size.
- Due to the single inflation line, the proximal cuff fills first stabilizing the tube according to the anatomy of the patient, the distal cuff then inflates automatically.

Mask size	Maximum cuff volume (mL)
0	10
1	20
2	35
3	60
4	80
5	90

The LT is well-tolerated until the return of the protective reflexes. Slight cuff deflation at this point allows better toleration of the oropharyngeal cuff.

- The device should be removed with the patient either deeply anesthetized or totally awake, otherwise laryngospasm, coughing or gagging may occur.
- Before removal of the device the cuffs should be completely deflated. Inadequate cuff deflation can make removal difficult, risking cuff damage, and discomfort for the patient.

Indications and Advantages

- The slim profile of the LT allows easy insertion with small mouth opening; thus, it can be considered for airway management in patients with restricted mouth opening.
- In difficult airway situation, it can be used as an emergency device to secure airway.

Laryngeal tube as a guide to endotracheal intubation: Tracheal intubation can be done blindly through the LT using a small size ETT (up to 6.5 mm). The ETT should be well-lubricated and the connector removed. If there is any resistance it is likely to be secondary to incorrect positioning of the LT, obstruction by the epiglottis. After correct placement of ETT, the LT cuffs should be deflated and the device can then be removed. The diameter of the ETT is limited by the LT size. Removing the LT over the ETT can be problematic, so in such a situation when a larger diameter ETT is desired, tube exchanger can be used. ET intubation can also be performed fiberoptically using airway exchange catheter.

Q. Discuss the cuffed pharyngeal sealer—Cobra PLA™.

Ans. The Cobra PLA™ (Pulmodyne®) (**Fig. 15**) consists of a breathing tube with a distal circumferential inflatable cuff proximal to the ventilation outlet. It is named so because of the widened distal part resembles the hood of the Cobra. This distal part prevents soft tissue collapse and allows positive pressure ventilation. When properly inserted, the Cobra head sits over glottis and seals off the hypopharynx. Tip of the Cobra PLA lies proximal to the esophageal inlet unlike other SGAs. A ramp present within this head, which directs the ventilation or a blindly passed ETT into the glottic aperture. A soft flexible grill placed anteriorly prevents epiglottis from downfolding. The grill allows passage of an ETT when required. It has a circumferential cuff which rests in the hypopharynx at the vallecula. When inflated, it lifts

the base of the tongue exposing the glottic aperture, and also seals the airway. The Cobra PLA is available in eight sizes according to the weight and size of the patient including adult and pediatric.

Cobra PLA sizes according to weight of the patient:

Size	Weight (kg)
½	2.5–7.5
1	5–15
1 ½	10–35
2	20–60
3	40–100
4	70–130
5	100–160
6	>130

Cobra PLA cuff inflation volumes suggested by manufacturer:

Size	Volume (mL)
3	<65
4	<70
5	<85

Insertion

- Deflate the cuff completely and fold it back against the breathing tube.
- Generously lubricate front and the back of the Cobra head and the cuff, but take care not to obstruct the grill.
- Position the patient's head in the sniffing position and open the mouth with the left hand.
- Direct the distal end of the Cobra PLA between the tongue and hard palate and not the hard palate. Once fully inserted in the mouth, perform a jaw lift and insert the Cobra PLA.
- Pushing the jaw downward makes insertion more difficult. Slight neck extension may aid passage of the device, as it turns toward the glottis at the back of the mouth.
- When positioned properly, the flexible tip is situated below the arytenoids, the cuff lies in the hypopharynx at the vallecula and the ramp and grill elevate the epiglottis.
- Once you are happy that Cobra PLA is properly inserted, inflate the cuff. Do not use the maximum volume advocated by the manufacturers. Rather inflate slowly while ventilating so that you get no leak. This is called the minimal leakage technique.
- Confirm correct placement by gentle positive pressure ventilation (peak airway pressure not >25 cm H₂O).
- Measure and document the pressure at which an audible leak is heard.



Fig. 15: Cobra PLA.

Removal

Once the patient becomes conscious deflate the cuff partially and gently withdraw the device from the mouth. This helps in removing collected secretions from patient's oral cavity.

Size of ETT that can pass through various sizes of Cobra PLA:

Cobra size	ETT size (mm)
½	3.0
1	4.5
1½	4.5
2	6.5
3	6.5
4	8.0
5	8.0
6	8.0

(ETT: endotracheal tube).

Q. Discuss the cuffless preshaped sealer—streamlined liner of the pharynx airway (SLIPA™).

Ans. The SLIPA™ is a latex-free device and it is shaped like a slipper (**Fig. 16**). It is preformed airway that is a cuffless and lines the pharynx when in place. It is made of medical grade thermoplastic material that is stiff enough to facilitate easy insertion. After insertion, it warms up to body temperature and softens, improving the seal and comfort. It has a hollow chamber similar to a boot in shape. There is a toe which sits at the upper end of esophageal opening, a bridge that comes to rest at the vallecula. Finally, the heel of SLIPA lends stability to the device by fitting in the nasopharynx. The bridge in the center of the chamber has two lateral bulges: these bulges occupy the piriform fossa of the larynx. These prevent downfolding of the epiglottis in front of glottic opening.

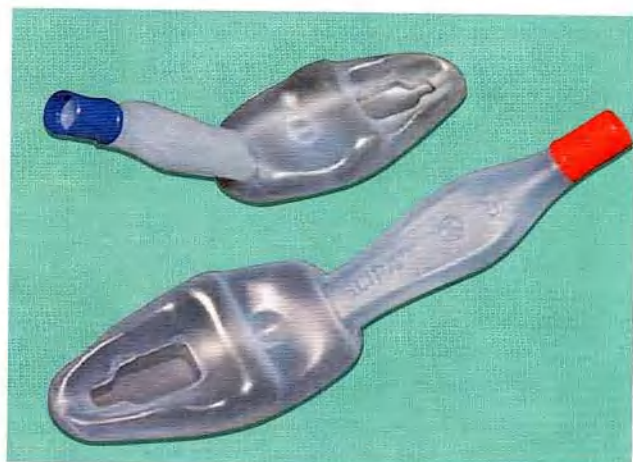


Fig. 16: Streamlined liner of the pharynx airway (SLIPA).

The chamber has a variable capacity (maximal of 50 mL SLIPA of size 53). This can store secretions and gastric contents, preventing their aspiration in the airway. It is available in six adult sizes: 47, 49, 51, 53, 55, and 57. The numbers correspond to the maximal transverse diameter (in mm) of the bridge. The best way of selecting SLIPA of the correct size is to see the distance between two cornu of the thyroid cartilage and selecting a size that will match the width of the bridge.

Patient size	SLIPA size	Median height	Height range
Very small female	47	152 cm (5'0")	145–160 cm (4'9"–5'3")
Small female	49	160 cm (5'3")	152–168 cm (5'0"–5'6")
Medium female	51	168 cm (5'6")	160–175 cm (5'3"–5'9")
Large female–small male	53	173 cm (5'8")	163–182 cm (5'4"–6'0")
Medium male	55	182 cm (6'0")	173–193 cm (5'8"–6'4")
Large male	57	190 cm (6'3")	180–200 cm (5'11"–6'7")

(SLIPA: streamlined liner of the pharynx airway).

Note: If unsure between two sizes, choose the larger size.

Insertion Technique and Device Removal

- Unlike LMA, we do not have to glide SLIPA along the hard palate. Its preformed curve allows the device to pass oropharyngeal curve easily.
- The head and neck are in sniffing position and after opening the mouth (and doing a forward jaw lift if required), the SLIPA is advanced toward the esophagus. The heel of the device automatically fits into the nasopharynx.
- The shape of the toe reduces the chances of airway obstruction, due to any reasons, i.e., either laryngospasm or downfolding of epiglottis.

Indications and Advantages

- The SLIPA is a cheap uncomplicated disposable device. The risk of aspiration is minimal with SLIPA.
- The SLIPA design has a large empty compartment, which has a space for collection of almost 50 mL oropharyngeal content, therefore the patient may be protected against aspiration of gastric content.
- Low cost compared to other SGAs.

Disadvantages

- There are no studies evaluating use of SLIPA used as conduit to fiberoptic bronchoscopy and intubation.
- Selecting the correct size is difficult.
- First attempts at insertion are successful in 76–85% patients.

Q. Discuss I-gel supraglottic airway.

Ans. I-gel is a soft, gel-like, cuffless SGA made of thermoplastic elastomer (**Fig. 17**). It creates a noninflatable anatomical seal of the pharyngeal, laryngeal and perilaryngeal structures. Because of its noninflatable seal it avoids compression trauma that can occur with inflatable SGA devices.

Design of I-gel, makes it an anatomical device as the distal soft noninflatable cuff achieves a mirrored impression of the pharyngeal, laryngeal, and perilaryngeal structures, and thus positions itself over laryngeal framework providing a reliable perilaryngeal seal. The tip of the distal noninflatable cuff lies in the proximal opening of the esophagus, isolating the esophageal opening from the laryngeal inlet. The proximal end of the cuff has an epiglottic rest with a ridge. The rest prevents the epiglottis from downfolding whereas the ridge is in contact with the base tongue preventing the upward and outward movement of the I-gel.

It consists of elliptical buccal cavity stabilizer which incorporates a circular airway lumen and a lumen for gastric tube insertion. The elliptical shape provides vertical stability and axial strength after insertion. Size 1 I-gel does not have a gastric lumen. It also has a built-in bite block incorporated within the tube. There is a horizontal line in the integrated bite block (in size 3, 4, and 5) which is guide for correct depth of insertion. If correctly inserted should coincide with the patient's teeth. The pediatric sizes do not have the horizontal line due to the greater variability in the length of the oropharyngeal-laryngeal arch in children.

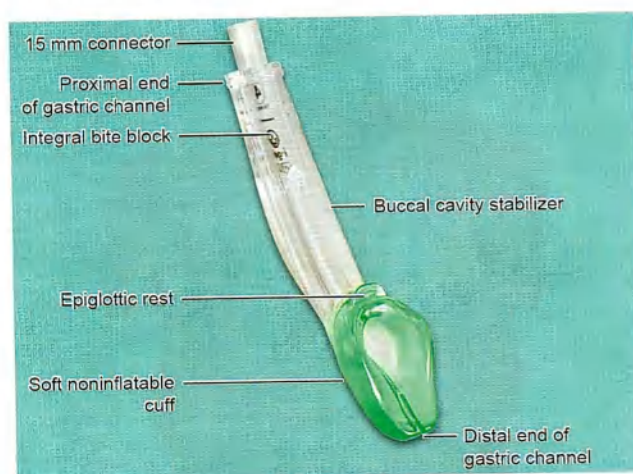


Fig. 17: I-gel supraglottic airway.

Advantages

- Provides minimal risk of tissue compression because of the noninflatable cuff in comparison to other SGAs.
- Latex free, sterile, single patient-use device.

I-gel size	Patient size	Patient weight (kg)
1	Neonate	2–5
1.5	Infant	5–12
2	Small pediatric	10–25
2.5	Large pediatric	25–35
3	Small adult	30–60
4	Medium adult	50–90
5	Large adult	90 +

Insertion Technique

- Lubricate the posterior aspect of the distal noninflatable cuff.
- Extend the patient head and flex the neck gently (sniffing the morning air position) then introduce the I-gel in the oral cavity with the concavity facing downward toward the chin.
- Glide the cuff along the palate until resistance is felt.
- The horizontal line should coincide with the patient's teeth, if it is not so then it is incompletely inserted. In such a case, removal the I-gel and reinsert it with jaw thrust. If this still does not solve the problem, then opt for a smaller size.
- Connect to the breathing apparatus.

Removal

- Once consciousness is regained and the protective reflexes are present, suction the oral cavity, and pharynx.
- I-gel can be removed when the patient is awake and easily arousable with verbal commands. Alternately, in patients with hyper-reactive airway [i.e., smokers, asthmatics or patients with chronic obstructive pulmonary disease (COPD)] can be removed in deeper plane of anesthesia.

Q. Describe the Elisha airway device (EAD).

Ans. The EAD is unique airway because of its ability to combine three functions, i.e., (1) ventilation, (2) intubation (blind and/or fiberoptic aided), and (3) gastric tube insertion. In comparison to the other SGAs EAD allows for simultaneous ventilation and intubation of the patient. It has three channels—ventilation, intubation, and gastric tube insertion (**Fig. 18**). The ventilation and the intubation ports are adjacent to each other whereas the gastric channel is on the posterior aspect of the device. The ventilation and intubation ports are separated at the proximal end of the device; however, they communicate with each other at the distal end into a laryngeal outlet. The ventilation channel has a 15 mm connector attached to the device. The intubation channel permits an 8.0 mm ID ETT for blind or fiberoptic-guided intubation.

The device consists of two high-volume low-pressure cuffs with a single inflation port. The proximal cuff seals

the nasopharynx and oropharynx, whereas the distal cuff seals the esophagus.

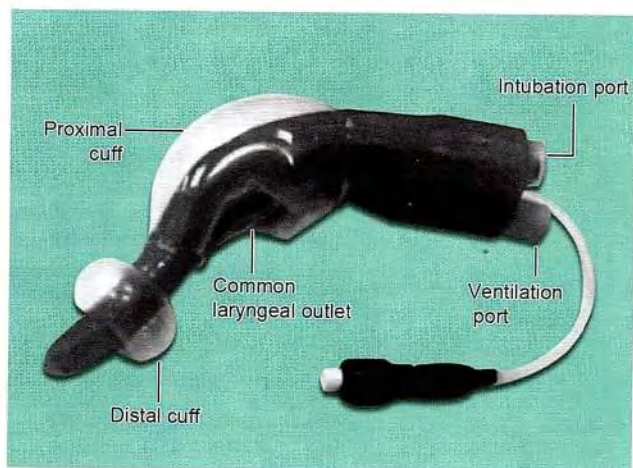


Fig. 18: Elisha airway device.

Q. Describe the Baska Mask.

Ans. The Baska Mask® (Logikal Health Products Pvt Ltd., Morisset, NSW, Australia) is a novel SGA device similar to

the LMA (Figs. 19 and 20). It has a noninflatable cuff, thus avoiding the risk of overinflation of cuff. The expandable, membranous cuff is continuous with the airway channel and thus gets inflated with positive pressure ventilation and produces effective seal, reducing chances of gastric insufflation, and aspiration.

The Baska Mask has an opening that sits in the upper esophagus and the dorsal surface of the cuff is made in a way that it directs any oropharyngeal contents such as secretions toward the side channels to which suction can be attached. It comes with an elbow connector for suctioning. The tube also has an in-built bite block, reducing the risk of patients' biting the airway. It has a tab attached in the mid-shaft of the tube, pulling the tab causes the shaft to bend like a preformed airway masks which may be used in patients with limited extension during insertion of the device. Anteriorly to the cuff, there is a hand-tab attached which allows manipulation during insertion, which is done in the neutral position. At the proximal end there are loops which help in fixation of the mask. It comes in four sizes that have color coded 15 mm connectors.

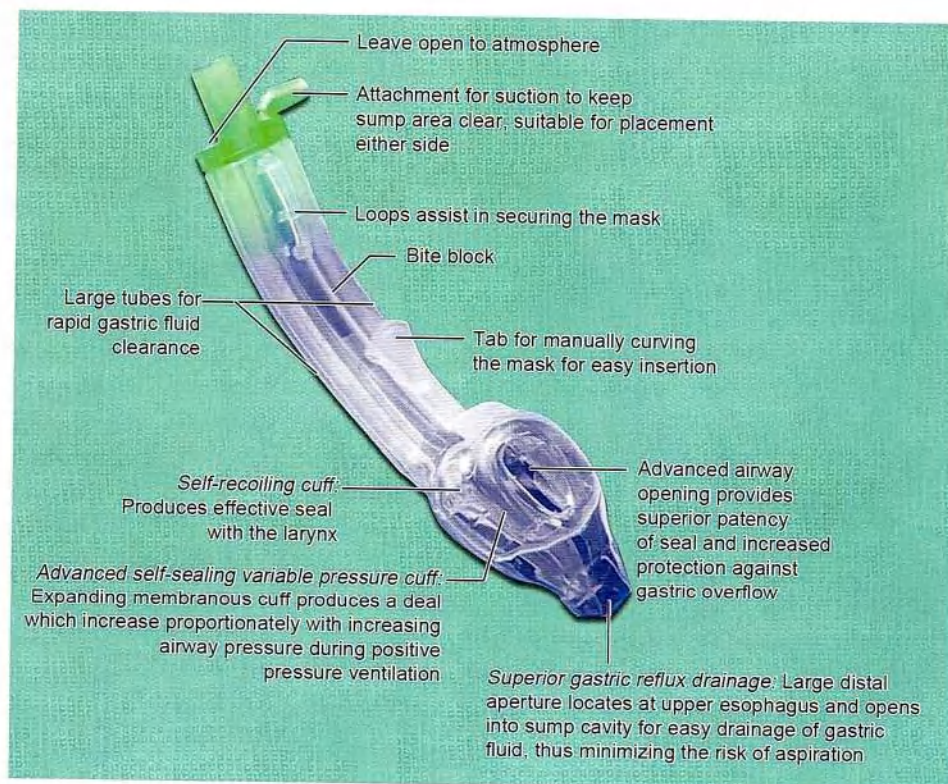


Fig. 19: The Baska Mask®.



Fig. 20: The Baska Mask.

Q. Describe LMA[®] Protector[™] and LMA[®] Gastro[™] Airway.

Ans.

LMA Protector

The LMA[®] Protector[™] (Fig. 21) is a second-generation, anatomically curved, single use perilaryngeal-sealer type of supraglottic device. It has a large volume pharyngeal chamber with dual gastric drainage channels. It is designed to functionally separate respiratory and gastrointestinal systems. Dual gastric drainage channel has a male and a female port. Male port allows for suctioning directly at the upper esophageal sphincter. Alternatively, gastric tube can be inserted from the female port. The cuff and the airway tube are made of 100% silicone. The elongated cuff of this device helps in achieving a laryngeal seal due to elliptical airway tube and a second seal is formed at the

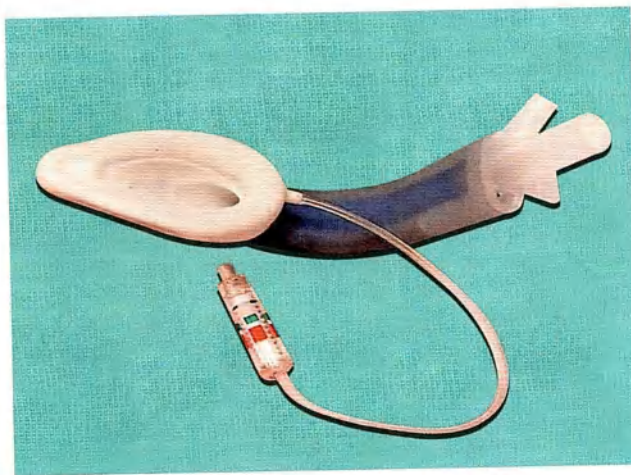


Fig. 21: Laryngeal mask airway (LMA) Protector.

upper esophageal sphincter. The cuff has a built-in pressure indicator which allows for continuous monitoring of cuff pressure. This integrated cuff pressure indicator reduces risks of over inflation (sore throat, dysphagia, hypoglossal, lingual, or recurrent laryngeal nerve palsies) and under inflation (increased risk of aspiration due to leakage around the cuff). It is available in sizes 3, 4, and 5. Size 3 allows ETT with 6.5 mm ID, whereas sizes 4 and 5 allow ETT with ID of 7.5 mm. The gastric drainage channel of size 3 LMA protector airway allows a 16 Fr gastric tube and size 3 and 4 can incorporate 18 Fr gastric tube.

Different sizes of LMA protector airway and maximum allowable ETT and orogastric tube sizes.

Size	Patient size	Maximum orogastric tube size	Maximum intracuff pressure	Maximum ETT tube size
3	30–50 kg	16 Fr	60 cm H ₂ O	6.5 mm
4	50–70 kg	18 Fr	60 cm H ₂ O	7.5 mm
5	70–100 kg	18 Fr	60 cm H ₂ O	7.5 mm

(ETT: endotracheal tube).

LMA[®] Gastro[™] Airway

The LMA Gastro airway is similar to the LMA Protector, however instead of the gastric drainage port it has a port for endoscope (Fig. 22). All available sizes of LMA Gastro allow a 14 mm endoscope, thus enabling anesthetists to control patient's airway and provide anesthesia simultaneously during endoscopic procedures. It is available in sizes 3, 4, and 5.

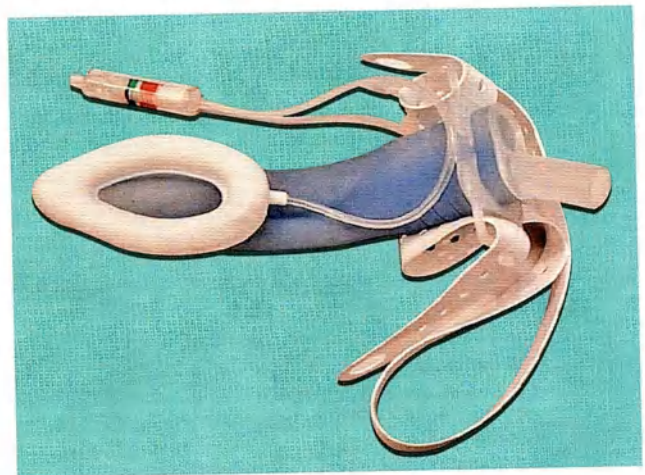


Fig. 22: Laryngeal mask airway (LMA) Gastro airway.

Q. Describe Blockbuster[™] laryngeal mask airway.

Ans. It is a second-generation supraglottic device with intubation capability and access for gastric drainage (Fig. 23). It is made up of silicone and the airway tube is

angulated more than $>95^\circ$ which gives it a shape similar to oropharyngeal curve. This makes intubation easier through this device. It has an integrated bite block and a four-way connector for fixing the LMA after insertion. The gastric outlet of Blockbuster LMA is bevel shaped allowing easier insertion of gastric tube. The mask portion of the LMA has a built-in guidance device which directs the ETT toward larynx at an angle of 30° . It has a reinforced ETT with a soft angulated tip flexible tip, which faces posteriorly. Blockbuster LMA is available in sizes 3, 4, and 5. Size 3 accepts 6.5–7.0 mm Blockbuster ETT (BETT), size 4 accepts 7.0–7.5 mm BETT, and 5 can take 7.5–8.0 mm BETT. Blockbuster LMA can be used for blind or flexible endoscopy-guided intubation.

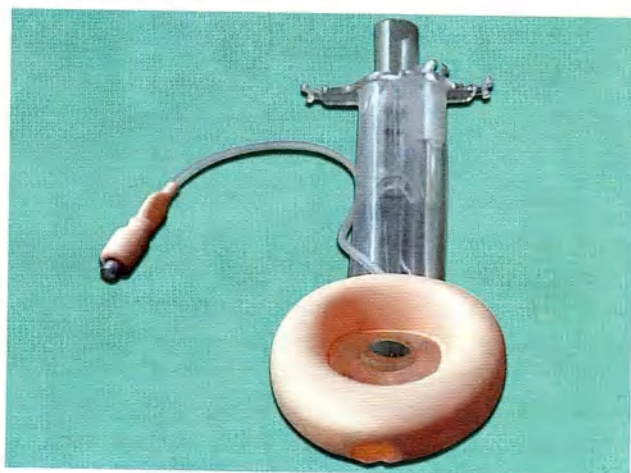


Fig. 23: Blockbuster laryngeal mask airway.

SUGGESTED READING

- Alexiev V, Salim A, Kevin LG, Laffey JG. An observational study of the Baska[®] mask: a novel supraglottic airway. *Anaesthesia*. 2012;67:640-5.
- Ambu. Ambu[®] AuraGain[™] Disposable Laryngeal Mask. [online] Available from <https://www.ambu.com/products/airway-management/laryngeal-masks/product/ambu-auragain-disposable-laryngeal-mask> [Last accessed March, 2020].
- Baska Mask. [online] Available from <https://baskamask.com.au/> [Last accessed March, 2020].
- Halwagi AE, Massicotte N, Lallo A, Gauthier A, Boudreault D, Ruel M, et al. Tracheal intubation through the I-gel[™] supraglottic airway versus the LMA Fastrach[™]: a randomized controlled trial. *Anesth Analg*. 2012;114:152-6.
- Hooshangi H, Wong DT. Brief review: the Cobra Perilaryngeal Airway [CobraPLA and the Streamlined Liner of Pharyngeal Airway (SLIPA)] supraglottic airways. *Can J Anaesth*. 2008;55: 177-85.
- <https://www.intersurgical.com/info/igel>
- <https://www.vbm-medical.com/products/airway-management/laryngeal-tubes/>
- Jeon YS, Choi JW, Jung HS, Kim YS, Kim DW, Kim JH, et al. Effect of continuous cuff pressure regulator in general anesthesia with laryngeal mask airway. *J Int Med Res*. 2011;39:1900-7.
- Ramachandran SK, Mathis MR, Tremper KK, Shanks AM, Kheterpal S. Predictors and clinical outcomes from failed Laryngeal Mask Airway Unique[™]: a study of 15,795 patients. *Anesthesiology*. 2012;116:1217-26.
- Seet E, Yousaf F, Gupta S, Subramanyam R, Wong DT, Chung F. Use of manometry for laryngeal mask airway reduces postoperative pharyngolaryngeal adverse events: a prospective, randomized trial. *Anesthesiology*. 2010;112: 652-7.
- Teleflex. (2016). LMA[®] Protector[™] Airway with Cuff Pilot[™] Technology. [online] Available from <https://www.lmaco.com/sites/default/files/34774%20LMA-Protector-Factsheet-INT-vis01r1-LR.pdf> [Last accessed March, 2020].
- Teleflex. (2018). LMA[®] Gastro[™] Airway with Cuff Pilot[™] Technology. [online] Available from <https://www.teleflex.com/la/en/product-areas/anesthesia/airway-management/laryngeal-masks/EN-LMA-Gastro-Data-Sheet.pdf> [Last accessed March, 2020].
- Tuoren. BlockBuster[™] Laryngeal Mask Airway. [online] Available from <http://tuoren.in/product/blockbuster-TM-laryngeal-mask-airway> [Last accessed March, 2020].
- Vaida SJ, Gaitini D, Ben-David B, Somri M, Hagberg CA, Gaitini LA. A new supraglottic airway, the Elisha airway device: a preliminary study. *Anesth Analg*. 2004;99:124-7.
- Vandse R, Kothari DS, Tripathi RS, Lopez L, Stawicki SP, Papadimos TJ. Negative pressure pulmonary edema with laryngeal mask airway use: recognition, pathophysiology and treatment modalities. *Int J Crit Illn Inj Sci*. 2012;2: 98-103.
- Zhang J, Zhao Z, Chen Y, Zhang X. New insights into the mechanism of injury to the recurrent laryngeal nerve associated with the laryngeal mask airway. *Med Sci Monit*. 2010;16:HY7-9.
- Zundert T, Gatt S. The Baska Mask[®]—A new concept in self-sealing membrane cuff extraglottic airway devices, using a sump and two gastric drains: a critical evaluation. *J Obstet Anaesth Crit Care*. 2012;2:23-30.

Regional Anesthesia Instruments

Parmanand Jain, Atul Prabhakar Kulkarni, Jeson Rajan Doctor

Q. What is the special advantage of using regional anesthesia?

Ans. Regional anesthesia techniques offer several advantages, most of which relate to postoperative recovery:

- Minimal or no emergence time
- Low or no incidence of postoperative nausea, vomiting, drowsiness, and dizziness provided deep sedation and narcotic use are avoided
- Minimal need for nursing care
- Eliminate the need for postoperative narcotics and its related side effects
- Facilitate rapid discharge from recovery and hospital.

Q. What is the Bromage scale of assessing the degree of motor block?

Ans. The Bromage scale of assessing motor block is given in **Table 1**.

TABLE 1: Bromage scale.

Scale	Criteria	Degree of block
0	Free movement of legs and feet able to raise extended leg	None
1	Inability to raise extended leg, knee flexion decreased, and full flexion of feet and ankles	Partial (33%)
2	Inability to raise leg or flex knees; flexion of ankle and feet present	Partial (66%)
3	Inability to raise leg, flex knee or ankle, or move toes	Complete

Q. What is the modified Bromage scale described by Breen?

Ans. The modified Bromage scale is described in **Table 2**.

TABLE 2: Modified Bromage scale.

Score	Criteria
1	Complete block (unable to move feet or knees)
2	Almost complete block (able to move feet only)
3	Partial block (just able to move knees)

Contd...

Score	Criteria
4	Detectable weakness of hip flexion while supine (full flexion of knees)
5	No detectable weakness of hip flexion while supine
6	Able to perform partial knee bend

Q. What is the desired level of anesthesia required for various surgeries?

Ans. Types of spinal analgesia (**Table 3**):

- **Saddle block:** Analgesia up to S1, usual puncture site between L4 and L5 or L5 and S1, cystoscopy, perineal, or anal surgery can be done.
- **Low spinal:** Analgesia up to L1 or T12, usual puncture site between L3 and L4, perineal or anal surgery, prostatectomy, etc.
- **Medium spinal:** Analgesia up to T10–T8, usual puncture site between L2 and L3 or between L3 and L4. Lower abdominal surgery can be done.
- **High spinal:** Analgesia up to T4, usual puncture site between L2 and L3, all abdominal surgeries can be done.
- **Unilateral:** Patient should be kept in lateral position after injection of analgesic solution for some minutes to fix the drug. It helps to minimize the sympathetic block and hemodynamic changes.

TABLE 3: Level of anesthesia required for various surgeries.

Level	Surgical procedure
T4–5 (nipple)	Upper abdominal surgery
T6–8 (xiphoid)	Intestinal (appendectomy, gynecologic, pelvic, and ureter and renal pelvic surgery)
T10 (umbilicus)	Transurethral resection, obstetric, and hip surgery
L1 (inguinal ligament)	Transurethral resection, and thigh and lower limb amputations
L2–3 (knee and below)	Foot surgery
S2–5 (perineal)	Perineal surgery, hemorrhoidectomy, and anal dilation

Contd...

Q. What is the Stout principle of spread of local anesthetic in spinal anesthesia?

Ans.

- Height of anesthesia varies directly with concentration of solution
- Extent of anesthesia is inversely proportional to rapidity of fixation
- Extent of anesthesia is directly proportional to the volume of drug used
- Extent of anesthesia is inversely proportional to spinal fluid pressure
- Extent of anesthesia is directly proportional to specific gravity for hyperbaric solution
- With isobaric or hypobaric solution extent depends on the position of patient.

Q. What is combined spinal-epidural anesthesia?

Ans. Combined spinal-epidural anesthesia is a technique in which the extradural space is located in the usual way as for extradural anesthesia. Then spinal anesthesia is performed either alongside or, more commonly, by inserting a long spinal needle through the extradural needle to puncture the dura. The advantages of spinal anesthesia (speed of onset and density of block) are therefore combined with those of extradural anesthesia (flexibility and the ability to extend for intraoperative or postoperative analgesia). In addition, the technique allows finer spinal needles to be used if a needle-through-needle method is employed, since the position of the needle tip can be estimated more easily as it passes through the extradural space into the dura. This technique has been widely used in obstetric analgesia and anesthesia and other types of surgery.

Techniques of needle insertion are as follows:

- *Single pass:* It is described by Soresi where an epidural injection was performed and then the same needle was advanced into the subarachnoid space.
- *Needle-through-needle:* The epidural space is identified as if performing an epidural block. A spinal needle is then passed through the epidural needle and beyond

its tip, until the dura is punctured. Subarachnoid drug is administered and after removing the spinal needle, an epidural catheter is placed. This technique requires that subarachnoid blockade is initiated before epidural catheter placement. Potential problems include failure of the spinal component, inadvertent insertion of the catheter into the subarachnoid space, and damage to either of the needles by friction between them.

- *Separate needles:* Separate interspaces require the placement of the epidural catheter first followed by the subarachnoid block.
- *Combined needle:* It is made by joining a spinal needle along the length of a Tuohy needle, including bending around the tip. They were designed to avoid friction between spinal and epidural needle and to ensure that dural puncture was separated from epidural catheter placement.

Q. Describe the various types of spinal/epidural needles.

Ans. Types of needles used for spinal and epidural blockade are described below (Fig. 1).

Spinal Needles

- The *standard spinal needle* consists of three parts: a hub that is fused to a cannula with a point, and a fitted removable stylet that occludes the distal lumen, and point of the cannula.
- Spinal needles are carefully manufactured without surface irregularities and with a tight-fitting removable stylet that completely occludes the needle lumen. A wide variety of sizes with different applications are available from 16 to 30 gauge.
- The points of the cannulae usually are made of stainless steel and must meet standards of stiffness, flexibility, and resistance to breakage. Luminal sizes of the spinal needle cannulae vary from 3.5 to 4 inches in length; smaller cannulae of 28 and 30 gauge are available. However, the "feel" for tissue during puncture is lost with these, and they are used, if at all, in neonates and infants.

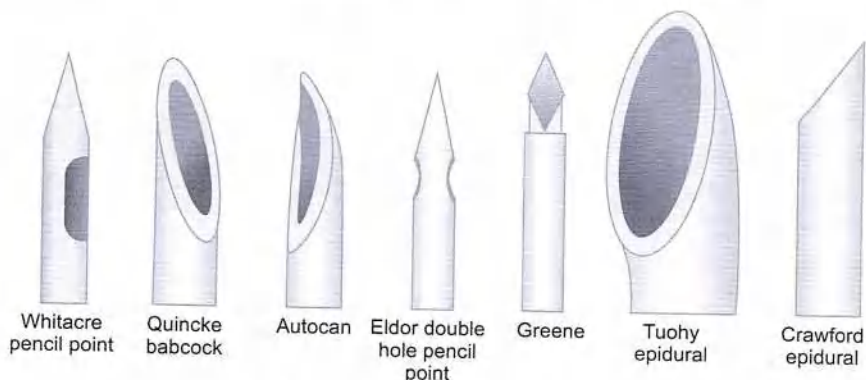


Fig. 1: Spinal and epidural needles.

- The needles are either sharp or blunt at the tip with either end-injection or side injection and either sharp or round (blunt) bevel edges. The *Quincke-Babcock* needle is considered the standard spinal needle. It has a small hub, medium bevel length with sharp edges, a sharp point, and end injection. There is a fitted stylet with a matching beveled tip to the cannula point. The hub is designed with a Luer lock-connector.
- The *Pitkin* needle has a short and sharp bevel with a pointed end injection. The stylet also has a short bevel which fits the point of the cannula.
- Two common alternatives are the *Greene* and *Whitacre* needles. The *Greene* needle has a small hub and a Luer lock connector. There are rounded, noncutting edges to the bevel, and the bevel is of medium length. There is also a stylet with a rounded bevel to fill the opening point of the cannula. This needle functions as a type of pencil point and because of the noncutting edges of the bevel, it separates rather than cut the dural fibers. Available sizes vary from 20 to 26 gauge and length ranges from 3 to 3.5 inches.
- The *Whitacre* and other *pencil-point* needles have a rounded bevel, no cutting edges, and the orifice of the needle is on one side of the cannula about 2.5 mm proximal to the tip of the cannula; a fitted stylet occludes the exit port on the side. The point cleanly separates all the fibers and the dural puncture site is small so that the incidence of postdural puncture headache (PDPH) is low because of less cerebrospinal fluid (CSF) leak. The factors affecting PDPH are as follows:
 - *Size of the needle:* A 25-gauge Quincke needle—less leakage than with a 22-gauge needle.
 - *Type of needle design:* Whitacre pencil point or Greene conical produces less leakage than needles that have a bevel with a cutting edge.
 - *Orientation of the bevel:* A 22-gauge needle parallel to longitudinal dural fibers produces less leakage than when it is perpendicular to the dural axis.
 - *Angle of approach to dural puncture:* Less leakage at an angle of 30° than at an angle of 60° or 90°.

Epidural Needles

- The standard epidural needle is typically 16–18 gauge, 3 inches long, and has a blunt bevel with a gentle curve of 15–30° at the tip. This blunt bevel and curve allow the needle to pass through the ligamentum flavum and abut against dura, pushing it away rather than penetrating it. This creates the negative pressure that identifies the epidural space. The most common version of this needle is referred to as the Tuohy needle and the curved tip is the Huber tip.
- *Crawford point needle:* It is short beveled at 40–45° with smooth edges. The distal end is open, but a well-fitted stylet closes the orifice for insertion. It is made of stainless

steel with a 2% molybdenum content which improves resistance to corrosion in these reusable needles.

- *Hustead needle:* It is a modified Tuohy needle with a rounded tip and a bevel opening which is located 2.7 mm from the tip.

Q. Describe the features of spinal/epidural catheters.

Ans. Teflon plastic tubing is the most widely used material. It is available in a radiopaque form and fitted with a fine wire stylet. Sizes vary from tubing with and outside the diameter of 0.99 mm permitting passage through an 18 gauge, thin wall, and directional Huber point needle to tubing with an outside diameter of 0.25 mm, which can pass through a 20- or 21-gauge needle. Some have single terminal opening, while others may have several lateral holes with or without a terminal opening. Preferred are the disposable, radiopaque Teflon catheters, 20 gauge by 90–100 cm, fitted with a fine wire stylet, and a syringe adaptor marked at 5, 10, 15, and 20 cm from the distal end. The catheter will pass through an 18-gauge thin wall needle. Stimulating catheters are available which use electrical current to produce muscle twitches as a marker for successful epidural placement.

Q. Discuss the factors affecting epidural and spinal drug spread.

Ans. This can be discussed under the following heads:

- *Patient factors:* The age, sex, weight, and height will determine the spread of the drug. High intra-abdominal pressure and change in the anatomy of the epidural space (pregnancy), anatomical deformities of spine, and volume of CSF will also affect the drug spread.
- *Technical factors:* Position of the patient, site of injection (level), type of needle used, and placement of intrathecal catheters will also affect the spread of the drug. Barbotage, previously thought to affect the spread, has been shown not to affect the spread of the drug. Rapid injection may cause the drug to spread more when hypobaric solutions are used.
- *Properties of the injected solution:* Baricity of the injectate—solutions are made hyperbaric by the addition of sugar. Higher volume will spread more. Larger dose or higher concentration will affect the height of block. Higher viscosity will increase the spread of the drug.

Q. What are special precautions taken in patients who are on anticoagulation therapy?

Ans. With the twice-daily US dosing regimen of low-molecular-weight heparin, the first dose is usually given 12–24 hours after the surgery, and removal of the epidural catheter is recommended before initiation of thromboprophylaxis. If the epidural catheter is left in place during thromboprophylaxis with the twice-daily low-molecular-weight heparin dose, a 24-hour delay between the last heparin dose and removal of the epidural catheter is

recommended. Combination with other drugs influencing the coagulation system including prophylactic heparin may be dangerous. Similar considerations apply to spinal or epidural catheter removal (**Table 4**).

Mechanical methods of deep vein thrombosis (DVT) prophylaxis (e.g., intermittent calf compression boots or foot pumps) are effective alternatives in any situation where there is a wish to avoid pharmacological methods completely.

TABLE 4: Summary of ASRA 2018 guidelines for regional anesthesia in a patient receiving antithrombotic or thrombolytic therapy.

	Minimum delay between last dose and placement or removal of epidural catheter	Minimum delay after placement or removal of epidural catheter and subsequent dosing
Heparin		
• No contraindication • The risk of neuraxial bleeding may be increased in debilitated patients after prolonged therapy • Patients receiving heparin for greater than 4 days should have a platelet count assessed prior to neuraxial block and catheter removal [to rule out heparin-induced thrombocytopenia (HIT)]		
IV unfractionated heparin	4–6 hours	1 hour
SC unfractionated Heparin BD/TID dosage		
Low dose (5,000 U BD/TID)	4–6 hours	1 hour
High dose (<20,000 U/Day)	12 hours	Assess risk–benefit ratio
Therapeutic dose (>20,000 U/day)	24 hours	Assess risk–benefit ratio
Full anticoagulation of cardiac surgery	Currently, insufficient data and experience. Postoperative monitoring of neurologic function and selection of neuraxial solutions that minimize sensory and motor block is recommended to facilitate detection of new/progressive neurodeficits	
Low-molecular-weight heparin (LMWH) e.g., dalteparin, enoxaparin, and tinzaparin		
Twice-daily dosing	24 hours	• 4 hours • Avoid indwelling catheters during initiation of prophylactic or therapeutic BD dosing
Single-daily dosing	12 hours	• 4 hours • Postoperation dose 12 hours after epidural placement with catheter in situ

Contd...

	Minimum delay between last dose and placement or removal of epidural catheter	Minimum delay after placement or removal of epidural catheter and subsequent dosing
Traumatic needle or catheter placement may signify an increased risk of spinal hematoma, and it is recommended that this consideration be discussed with the surgeon. It does not necessitate postponement of surgery. However, initiation of LMWH therapy in this setting should be delayed for 24 hours postoperatively. - Routine use of monitoring of the anti-factor Xa level is not recommended - Antiplatelet or oral anticoagulant medications administered in combination with LMWH increase the risk of spinal hematoma - Patients receiving LMWH for greater than 4 days should have a platelet count assessed prior to neuraxial block or catheter removal to rule out HIT		
Warfarin		
- Stop drug at least 5 days prior to procedure - Bridge with heparin infusion if continued anticoagulation required - Check INR on day of procedure and ensure <1.4 INR units		
If INR >1.4:		
- Delay surgery - Consider alternative anesthetic/analgesic technique, if surgery urgent - Consider reversal with factor IX concentrate, FFP, and Vitamin K in an emergency (3-factor and 4-factor prothrombin complex concentrates)		
If epidural is to be removed, INR <1.4, PTT must be corrected with FFP or Vitamin K. Consider removal of neuraxial catheter prior to starting oral warfarin.		
Fibrinolytics/Thrombolytics (alteplase, reteplase, tenecteplase, urokinase, and streptokinase)		
Absolute contraindication for neuraxial blockade:		
- Thrombolytics drugs are contraindicated for 10 days following puncture of noncompressible vessels - In patients who have received fibrinolytic and thrombolytic drugs, we recommend against performance of spinal or epidural anesthetics		
Antiplatelet drugs		
Aspirin, NSAIDs, and COX-2 inhibitors	0 day	Immediately
In cases where aspirin is given in combination with other anti-coagulants, the risk of bleeding increases and the performance of neuraxial blockade should be reconsidered.		
Antiplatelet drugs (P2Y₁₂ receptor inhibitors)		
Clopidogrel	7 days	Daily maintenance dose—take 6 hours later, loading dose 24 hours later
Ticlopidine (rarely used now)	10 days (10–14 days)	6 hours
Prasugrel	7–10 days	6 hours
Ticagrelor	5–7 days	6 hours

Contd...

Contd...

Contd...

	Minimum delay between last dose and placement or removal of epidural catheter	Minimum delay after placement or removal of epidural catheter and subsequent dosing
Cangrelor	3 hours	8 hours
Antiplatelet drugs (GP IIb/IIIa antagonists) AVOID due to platelet dysfunction		
Abciximab	24–48 hours	AVOID (if necessary monitor neurological function)
Tirofiban	4–8 hours	AVOID (if necessary monitor neurological function)
Eptifibatide	4–8 hours	AVOID (if necessary monitor neurological function)
Other antiplatelet agents		
Dipyridamole	24 hours	6 hours
Cilostazol	2 days	6 hours
Antifactor Xa—fondaparinux		
Fondaparinux	36 hours	12 hours
Fondaparinux is recommended for thromboprophylaxis only with single needle pass, atraumatic needle placement, and avoidance of indwelling neuraxial catheters. If this is not feasible, an alternate method of prophylaxis should be considered.		
Newer oral anticoagulants (NOAC—factor Xa inhibitors)		
Rivaroxaban	- Placement—72 hours - Removal—22–26 hours	6 hours
Apixaban	- Placement—72 hours - Removal—26–30 hours	6 hours
Edoxaban	- Placement—72 hours - Removal—20–28 hours	6 hours
Betrixaban	- Placement and removal—72 hours - Avoid neuraxial block if CrCl <30 mL/min	5 hours
Direct thrombin inhibitors		
Parenteral, e.g., desirudin, bivalirudin, argatroban	AVOID	
Oral dabigatran	- Placement—120 hours - CrCl >80 mL/min—72 hours - CrCl: 50–79 mL/min—96 hours - CrCl: 30–50 mL/min—120 hours - CrCl <30 mL/min—AVOID	6 hours

Contd...

Contd...

	Minimum delay between last dose and placement or removal of epidural catheter	Minimum delay after placement or removal of epidural catheter and subsequent dosing
Herbal remedies (check for concomitant other blood thinners)		
Garlic	Stop 1 week prior, also stop other antiplatelet agents	
Dong quai/ Danshen	Check INR with warfarin	
Ginkgo Biloba	Check platelet function	
SSRIs	Caution in patients on SSRIs	

(ASRA: American Society of Regional Anesthesia; SSRIs: selective serotonin reuptake inhibitors; COX-2: cyclooxygenase-2; INR: international normalized ratio; FFP: fresh frozen plasma; NSAID: nonsteroidal anti-inflammatory drug; PTT: partial thromboplastin time).

SUGGESTED READING

- Breen TW, Shapiro T, Glass B, Foster-Payne D, Oriol NE. Epidural anesthesia for labor in an ambulatory patient. *Anesth Analg*. 1993;77(5):919-24.
- Calthorpe N. The history of spinal needles: getting to the point. *Anesthesia*. 2004;59(12):1231-41.
- Denny NM, Selander DE. Continuous spinal anesthesia. *Br J Anaesth*. 1998;81:590-7.
- Di Cianni S, Rossi M, Casati A, Cocco C, Fanelli G. Spinal anesthesia: an evergreen technique. *Acta Biomed*. 2008;79(1):9-17.
- Fettes PD, Jansson JR, Wildsmith JA. Failed spinal anesthesia: mechanisms, management, and prevention. *Br J Anaesth*. 2009;102(6):739-48.
- Hocking G, Wildsmith JA. Intrathecal drug spread. *Br J Anaesth*. 2004;93(4):568-78.
- Horlocker TT, Vandermeulen E, Kopp SL, Gogarten W, Leffert LR, Benzon HT. Regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy: American Society of Regional Anesthesia and Pain Medicine Evidence-Based Guidelines (Fourth Edition). *Reg Anesth Pain Med*. 2018;43(3):263-309.
- Lambert DH, Hurley RJ, Hertwig L, Datta S. Role of needle gauge and tip configuration in the production of lumbar puncture headache. *Reg Anesth*. 1997;22(1):66-72.
- Narouze S, Benzon HT, Provenzano DA, Buvanendran A, De Andres J, Deer TR, et al. Interventional spine and pain procedures in patients on antiplatelet and anticoagulant medications: guidelines from the American Society of Regional Anesthesia and Pain Medicine, the European Society of Regional Anaesthesia and Pain Therapy, the American Academy of Pain Medicine, the International Neuromodulation Society, the North American Neuromodulation Society, and the World Institute of Pain. *Reg Anesth Pain Med*. 2015;40(3):182-212.
- Stannard D. Epidural blood patching for preventing and treating postdural puncture headache. *J Perianesth Nurs*. 2011;26(6):411-2.
- Tsen LC, Hepner DL. Needles used for spinal anesthesia. *Expert Rev Med Devices*. 2006;3(4):499-508.

Nerve Locator and Peripheral Nerve Stimulator

Nayana Amin, Sudivya Sharma

Q. What is the difference between a nerve locator and a peripheral nerve stimulator (PNS)?

Ans. Peripheral nerve stimulator is used to assess the degree of neuromuscular block. Needle or surface electrodes are used to stimulate a suitable peripheral nerve and the evoked response of the muscles innervated by the nerve is either recorded or palpated. Electrical nerve locators are used as an aid to peripheral nerve blockade. Specialized insulated needles are used to elicit a response from a motor nerve. Both are battery-operated equipment. However, an electrical nerve locator can deliver currents in the range of 0.1–5 mA, while a peripheral nerve locator can deliver currents in the range of 10–100 mA. When using needle electrodes, lesser current is needed to stimulate a nerve due to close proximity to the nerve. While using surface electrodes, higher currents are needed as skin resistance has to be overcome. Also, in a PNS, different patterns of stimulation [train-of-four (TOF), double-burst stimulation (DBS), and post-tetanic count (PTC)] are available.

Q. What are the advantages of a nerve locator?

Ans. A nerve locator is helpful because:

- It has a higher success rate.
- It is easier to locate nerve—not totally dependent on anatomy.
- There is better localization of nerve trunks in difficult situations.
- There are less chances of nerve damage.
- There is no discomfort or pain to the patient.
- Patient cooperation is not required.
- It can be used on anesthetized nonparalyzed patients.
- Toxic dose of local anesthetic can be avoided by giving titrated volume.
- It is a valuable tool for training purpose.
- Blocks can be performed in pain clinics and rehabilitation centers.

Q. What are the limitations of a nerve locator?

Ans. A nerve locator has the following limitations:

- It is only applicable to peripheral nerves (not relevant to central axis blockade).
- The aim is to stimulate motor nerves; hence, its use is limited to mixed peripheral nerves. Its use has been described for pure sensory nerves, but it is not very common in clinical practice.
- Equipment costs.
- It cannot be used after paralysis with neuromuscular blocking drugs.
- PNS should not be used as a substitute for the lack of anatomical knowledge of the nerves that are being blocked.

Q. What do you understand by chronaxy and rheobase?

Ans. The amount of electrical energy required to propagate a nerve impulse is a product of the stimulus strength (mA) and duration of current (ms). Thus, stimulation of a nerve depends on the relationship between the strength and the duration of current flow. This relationship can be explained by the terms, rheobase and chronaxy. Rheobase is the minimum current strength required to generate an action potential. For any nerve type, there is a minimum current strength below which an impulse will not be generated, no matter for how long the current is applied. The chronaxy is the length of time the current has to be applied to the nerve to generate an action potential when the current strength is twice the rheobase.

Q. Why is stimulation of a peripheral nerve not painful for the patient?

Ans. As chronaxy varies in different nerve fibers, it is useful for comparing different nerves. Myelinated fibers have a shorter chronaxy than unmyelinated nerve fibers and require less electrical energy for stimulation. Since the large motor fibers ($A\alpha$) have a shorter chronaxy (0.05–0.1 ms),

they can be stimulated with pulses of current with a shorter width. Thus, when stimulating a motor nerve, the nerve fibers responsible for pain do not get stimulated as they have a longer chronaxy (A δ fibers have a chronaxy of 0.150 ms and C fibers have a chronaxy of 0.4 ms). As a result of this, the patient does not feel any pain during peripheral nerve blocks (PNBs) except when the needle is piercing the skin or is touching the nerve.

In an awake state, a cooperative patient's sensory nerves can be located by using longer width pulses of current.

Q. What is the importance of threshold current and what should be the threshold current for stimulating a nerve?

Ans. If the needle tip is further away from the nerve, then more current strength is required at the tip of needle to stimulate the nerve. According to Coulomb's law, $E = K(Q/r^2)$, where E is the threshold current required at the nerve, K is a constant, Q is the minimal current from the needle tip, and r is the distance from the nerve. Therefore, current required to stimulate a nerve is inversely proportional to the square of the distance. This principle is used to calculate the distance from the needle to the nerve using a constant current stimulus. For example, if the nerve is getting stimulated with the current threshold of 1 mA and the distance of nerve from tip of needle is 1 cm, then if the distance increases to 2 cm, the current required will be 4 mA and for a distance of 0.5 cm, the current will be 0.25 mA. If the needle is too far away, then stimulation of the nerve with higher current strengths can be very painful. Also, if the nerve can be stimulated with a lower threshold current, then it indicates that the needle tip is very close to the nerve. If muscle twitch is elicited with a current of 0.3–0.4 mA, it indicates that the needle tip is very close to the nerve and injection of local anesthetic at this point will result in a successful block. However, if muscle twitch is elicited with a current strength of less than 0.2 mA, there is a very strong possibility that the needle has penetrated the epineurium. Injection of local anesthetic at this point increases the risk of intraneural injection which may cause temporary or permanent neural damage.

Q. Will the polarity of the stimulating electrode make any difference during the use of a nerve locator?

Ans. If the stimulating needle is the cathode (negatively charged), then the flow of current alters the resting membrane potential of the surrounding cells. This produces an area of depolarization, making it easier to stimulate the nerve. Thus, significantly less current is required to stimulate the nerve if the stimulating electrode is a cathode. If the stimulating electrode is the anode (positively charged), then the flow of current causes an area of hyperpolarization adjacent to the tip of the needle and an

area of depolarization just distal to the tip. This arrangement is not very efficient and very high current strength is required for stimulating the nerve. In most modern PNSs, the needle is negative by default and it cannot be changed by the operator.

Q. What is the significance of the stimulus frequency during the use of a nerve locator?

Ans. The muscle response produced by a stimulating needle indicates that the needle is approaching the target nerve. If the needle is being advanced very fast and the stimulus frequency is very low, then the needle may either miss the nerve or it may go through the nerve resulting in damage to the nerve. However, if the stimulus frequency is very high, it can be very painful for the patient. In clinical practice, a frequency of 2 Hz is adequate and does not cause patient discomfort. If you are getting a motor response at a current strength of less than 1 mA, it indicates that the needle is very close to the nerve and then it should be advanced slowly at a speed of 1 mm/s so that you do not miss the nerve.

Q. Can you describe the parts of an electrical nerve locator? What is the importance of each part?

Ans. Electrical nerve locator has various parts that help to improve the efficiency and safety of the nerve locator.

Current generator produces the electric current required to stimulate the nerve.

Constant current output: The set current is delivered between the tip of the needle and the remote electrode. Modern electrical nerve locators compensate for the changes in resistance and ensure that a constant current is delivered. The resistance in the circuit will vary with position of the electrode from the tip of the needle and the tissue impedance.

Current meter: The intensity of current with which the nerve is stimulated gives an idea about the approximate distance from the tip of the needle to the nerve. Hence, it is important to have an accurate idea of the intensity of current, which is used to stimulate the nerve. The current meter gives a digital display of the current delivered in the circuit. The maximum and minimum current delivered by most electrical nerve locators is 5 and 0.1 mA, respectively. For patients with neuropathy and sensory nerve stimulation, higher current output is needed.

Current output control: The operator or assistant can precisely control the amount of current passing through the circuit with the help of the current output control. In some modern nerve locators, the control knob can be easily sterilized by autoclaving. Foot-operated current intensity controllers can be used whenever there is no assistance available.

Frequency control: In most electrical nerve locators, the impulses are delivered at a frequency of 1 or 2 Hz. Needle manipulation is better with higher frequencies as there is a frequent feedback to the operator as the needle is being advanced. The frequency may have to be reduced if the patient experiences pain during stimulation.

Connection/disconnection indicator: This is an important safety feature. Machines may either have flashing lights or pulsating beeps, which indicate that the connections are intact. Loss of these signals indicates that the connections are lost and the current will not be delivered. The most common cause of circuit disconnection is incomplete connection and/or poor electrode contact. Current may not be delivered if there is loss of circuitry, unit malfunction, or battery failure.

Q. Can a PNS used for neuromuscular monitoring be used for PNBs?

Ans. Peripheral nerve stimulators used in assessing recovery of muscle relaxation should not be used in locating nerves for blockade because of the possibility of high intensity currents (sometimes >100 mA), causing neural damage at close approximation to the nerve.

Q. What is the significance of the pulse width?

Ans. As mentioned earlier, motor fibers have a shorter chronaxy while the pain fibers have a longer chronaxy. A pulse width of 0.05–0.1 ms will preferentially stimulate the motor fibers and not the pain fibers. A longer pulse width of >0.15 ms can be used for locating pure sensory nerves such as lateral femoral cutaneous nerve. Patients will complain of paresthesias along the distribution of the nerve.

Q. What are the important features of a stimulating needle?

Ans. For precise nerve location, specially designed thin insulated needles have to be used with the nerve locator. Due to current dispersion, larger current is required to stimulate a nerve with a noninsulated needle. Specialized needles are completely insulated with the exception of the bevel or the tip as a result of which lower currents are required to stimulate the nerve. Use of these needles increases the accuracy, but it may be more difficult to locate the nerve as the current density is focused at the tip. Accidental damage to the nerves can be further minimized by the use of needles with noncutting tip. Needles are available in varying lengths (commonly 50, 100, and 150 mm) and the needle selection would depend on the depth of tissue plane required for a particular block. If the nerve is superficial then a thin and short needle can be used and if the nerve is deep then a long needle with a larger diameter will be needed. It is easy to manipulate long needles with a larger diameter, but it

also increases the chances of tissue damage. To prevent current leakage, the needle has an insulated cable, which is connected to the cathode of the nerve locator. The needle also has an extension tubing which minimizes the chances of needle movement while injecting the local anesthetic. The extension tubing should be flushed to remove any air as it can cause patchy block.

Q. What are the local anesthetic doses for PNB?

Ans. Large volumes of local anesthetic would be needed to perform a PNB. Accidental intravascular injections of this large volume may cause central nervous system (CNS) and cardiac complications. Therefore, the appropriate drug/dose selection, monitoring, and availability of resuscitative drugs and equipment are essential while performing PNB. When a combination of local anesthetic is used, the toxicity would be additive. The concentration and volume of local anesthetic should be reduced in elderly (patients >65 years) and in American Society of Anesthesiologists (ASA) 3 patients with significant underlying cardiac/hepatic/renal disease.

Therapeutic dose limits of commonly used local anesthetic for PNB are as follows:

- Bupivacaine, L-bupivacaine, and ropivacaine—3–4 mg/kg
- Lidocaine with epinephrine—5–7 mg/kg
- Lidocaine without epinephrine—3–5 mg/kg.

Q. What is the appropriate evoked motor response for each PNB technique?

Ans. The appropriate evoked motor response for each PNB technique is given in **Table 1**.

TABLE 1: Evoked motor response (EMR) for various blocks.

Peripheral nerve block technique	Optimal EMR
Interscalene	Flexor—deltoid, biceps, and pectoralis major Extensor—triceps, brachioradialis, and wrist extensors
Deep cervical plexus	Rhomboids and shoulder girdle
Infraclavicular	Muscles of wrist and hand Radial—extension of wrist and fingers Median—flexion of wrist and fingers Ulnar—adduction of thumb/4th and 5th finger flexion
Femoral	Quadriceps—patellar snap
Sciatic	Inversion and plantar flexion

Q. How can you improve the success rate of a block?

Ans. To minimize the latency (onset of action) and improve the success of the block: The local anesthetic must be deposited as close to the nerve as possible. Appropriate end

points with a PNS can increase the success rate of the block. While performing an axillary block, the highest success rate is achieved with an evoked motor response of the radial nerve and with sciatic nerve block, evoked motor response of inversion is associated with shortest latency and highest success rate.

The success rate is highest when the desired evoked motor response is obtained with a stimulating current which is between 0.2 and 0.4 mA. If evoked motor response is obtained at currents >0.5 mA, it indicates that the needle tip is far from the nerve and it may result in failed block. If a stimulating current of <0.2 mA elicits an evoked motor response, then there is an increased risk of nerve damage due to the possibility of intraneural injection.

The needle should be immobile during the injection. Judicious selection of local anesthetic drug and concentration is described in **Table 2**.

TABLE 2: Local anesthetics for blocks.

Local anesthetic with epinephrine (1:300,000)	Latency (min) surgical anesthesia	Duration (hour) surgical anesthesia	Duration (hour) postoperative analgesia
Lidocaine 1.5–2%	10–20	2–3.5	3–5
Bupivacaine 0.5–0.625%	15–30	5–6	12–24
L-bupivacaine 0.5–0.625%	15–30	5–6	12–24
Ropivacaine 0.5%	10–20	3–4	10–15

Latency is longer with axillary and sciatic nerve blocks than with interscalene and femoral nerve blocks.

Q. Can you describe the technique of PNB?

Ans.

- Explain the procedure to the patient. Take a written informed consent.
- Check for history of bleeding disorder.
- Before performing a PNB, take an IV access and ensure that the resuscitation drugs and airway equipment are readily available. The patient should be monitored during the block.
- Test the function of the nerve stimulator. Failure to check for circuit completion increases potential for neural damage (especially when performing nerve blocks in unconscious patients).
- Apply the electrode, ensuring good contact.
- Disinfect the skin, create a skin wheal and, if necessary, infiltrate the puncture channel.
- Connect the earth lead to the surface electrode. Connect the syringe containing local anesthetic to the injection line and flush it.

- Switch on the stimulator and select the pulse duration (0.1 ms for mixed nerves), pulse frequency (2 Hz), and stimulation current (1 mA).
- After inserting the needle into the skin and subcutaneous tissue, manipulate the needle to get the desired evoked motor response. Ask the assistant to reduce the stimulating current in small increments till the contractions are just barely visible at a threshold level of 0.2–0.3 mA. Evoked motor response at a current <0.2 mA indicates that the needle may be intraneural.
- If there are visible contractions at a threshold current <0.4 mA but not <0.2 mA, then inject 1 mL of local anesthetic after negative aspiration test. At this point, the twitching should disappear. If the patient experiences pain on injection or if the twitches do not disappear, then the needle should be withdrawn slightly as the needle may be intraneural.
- After a negative aspiration test, the rest of the local anesthetic is injected slowly and the needle is removed.
- Separate kits are available for continuous infusion techniques (e.g., contiplex).

Q. Are the PNS settings the same for any block?

Ans. No, the PNS settings change depending on the patient and the type of nerve to be stimulated:

- *Mixed nerve (most PNB) blocks:* Current—1 mA, current duration—0.1 ms, and frequency—1–2 Hz.
- *Sensory nerve (e.g., saphenous nerve) block:* Current—2–5 mA, current duration—1 ms, and frequency—1 Hz.
- *Diabetic neuropathy:* Current—2 mA, current duration—0.3 ms, and frequency—1–2 Hz.

Q. How does one avoid an intraneural injection?

Ans. While it is important to place local anesthetic accurately, it is vital to minimize the chance of causing nerve damage. The following list of seven points may help to avoid intraneural injection.

Equipment checks—check the integrity of the circuit and whether the device is delivering the current. Complete the circuit by connecting the negative and positive electrodes and switch on the nerve stimulator. There will be an audible beep if the current is being delivered. If there is no audible beep, it indicates that current is not being delivered and the device should not be used:

- Appropriate anatomical knowledge.
- Threshold—no muscle twitch at or below 0.2 mA.
- Twitch should disappear immediately after injection starts.
- Minimal resistance to injection.
- Patient should be awake to see if there is pain on injection.
- Avoid multiple attempts at nerve location.

Q. Why does the twitch disappear immediately on injection of local anesthetic?

Ans. Immediate disappearance of the twitch on injection local anesthetic is due to the mechanical displacement of the nerve away from the needle tip.

Q. What are the contraindications to PNBs?

Ans. **Absolute contraindication:** Patient refusal, infection at the site of block, allergy to local anesthetics, and severe coagulopathy.

Relative contraindication: Mild coagulopathy and distorted local anatomy (can consider blocks under ultrasound guidance along with a nerve stimulator).

Q. What are the motor supply areas of the upper and lower limbs?

Ans. The motor supply areas of the upper and lower limbs are described in **Tables 3 and 4**.

TABLE 3: Motor supply areas of upper limb.

Peripheral nerve	Muscle	Function
Axillary nerve	Deltoid muscle	Abduction of the arm in the shoulder joint
Musculocutaneous nerve	- Biceps brachii muscle - Coracobrachialis muscle	Bends the elbow in supination
Median nerve	Flexor carpi radialis muscle	Flexes and abducts the wrist radially
	Flexor pollicis brevis muscle	Pronates the forearm (flexes proximal phalanx of thumb)
	Flexor digitorum profundus	Flexes and adducts the thumb, flexes muscle (I–III) and fingers I–III (flexes distal interphalangeal joint of fingers)
Radial nerve	Triceps brachii muscle	Extends elbow
	Extensor carpi radialis (brevis) muscle	Extends and abducts the wrist radially
	Extensor digitorum muscle	Extends and flexes the hand dorsally and extends and spreads the fingers
Ulnar nerve	Flexor carpi ulnaris muscle	Flexes and abducts the wrist in an ulnar direction
	Flexor digitorum profundus	Flexes the fingers (IV–V) and the muscle (IV–V)

TABLE 4: Motor supply areas of lower limbs.

Peripheral nerve	Muscle	Function
Femoral nerve	Quadriceps femoris muscle	Flexes the hip and extends the knee
Obturator nerve	Adductors of thigh at the hip joint	Adducts the thigh

Contd...

Contd...

Peripheral nerve	Muscle	Function
Tibial nerve	Biceps femoris muscle	Flexes the knee and rotates the leg laterally
	Semimembranosus muscle	Flexes the knee
	Semitendinosus muscle	Extends the thigh, flexes the leg and rotates it medially
	Flexor hallucis longus muscle	Flexes the foot
	Flexor digitorum longus muscle	Flexes the toes
Common peroneal nerve	Tibialis anterior muscle	Dorsiflexion and inversion of the foot
	Extensor digitorum muscles, extensor hallucis muscles, and peroneal muscles	Extend, evert, and pronate the outer foot

Q. How can you further improve the safety and success of PNBs?

Ans. Performing the blocks under ultrasound guidance can further improve the safety and success rate of PNBs.

Q. Describe various PNBs.

Ans. The various PNBs are described in **Tables 5 to 9**.

TABLE 5: Interscalene block.

Indications	Local anesthetic drug and doses	Duration of analgesia	Potential problems	Contraindications
Shoulder arthroscopy, reconstructive shoulder surgery, and upper arm surgery	Bupivacaine 0.5% with epinephrine 1:200,000, volume 35–40 mL	12–18 hours	Common: Phrenic nerve block (inevitable), Horner syndrome, and recurrent laryngeal nerve block (hoarseness) Rare: Intravascular injection (vertebral artery), pneumothorax, and inadvertent spinal and epidural block	Patients with significant preexisting pulmonary disease

TABLE 6: Axillary block.

Indications	Local anesthetic drug and doses	Duration of analgesia	Potential problems
Elbow, forearm, and hand surgery	Bupivacaine 0.5% with epinephrine 1:200,000, volume 40–50 mL	12–24 hours	Hematoma, intravascular injection, and peripheral nerve injury

TABLE 7: Femoral nerve block (3-in-1 block).

Indications	Local anesthetic drug and doses	Duration of postoperative analgesia	Potential problems
Reconstructive knee procedures	Bupivacaine 0.5% with epinephrine 1:200,000, volume 20–30 mL	12–18 hours	Hematoma, intravascular injection, and quadriceps femoris weakness Patient should be prevented from bearing weight on the blocked extremity and instructed to crutch walking

TABLE 8: Sciatic nerve block/popliteal nerve block.

Indication	Local anesthetic drug and doses	Duration of postoperative analgesia	Potential problems
Surgical procedures below the knee	Bupivacaine 0.5% with epinephrine 1:200,000 volume 25–30 mL	Bupivacaine 12–24 hours	Incomplete block, inability to bear weight. Patient should be instructed to crutch walking

TABLE 9: Ankle block.

Indications	Local anesthetic drug and doses	Duration of postoperative analgesia	Potential problems
Surgery on foot	- Bupivacaine 0.5%, volume 30–40 mL - No epinephrine	Bupivacaine 12–24 hours	None

■ PERIPHERAL NERVE STIMULATOR

Q. Why do we need neuromuscular monitoring?

Ans. Traditionally clinical criteria were used to assess recovery from neuromuscular blockade. Numerous studies have shown that even if there is clinical recovery

of neuromuscular function, there is significant residual curarization in the postoperative recovery room. The residual curarization can lead to increased postoperative complications. The clinical signs of recovery from neuromuscular blockade can be influenced by various other factors other than the degree of neuromuscular blockade. Response of the muscle to nerve stimulation would give precise information about the status of the neuromuscular function.

During neuromuscular monitoring, a PNS is used to evaluate the muscular response to supramaximal stimulation of a peripheral motor nerve.

Q. What are the various ways in which a nerve can be stimulated? Which method of stimulation is commonly used and why?

Ans. A nerve can be stimulated by an electrical or a magnetic stimulus. Electrical nerve stimulation is commonly used in clinical practice. Unlike electrical nerve stimulation, magnetic nerve stimulation is less painful and does not require physical contact with the body. However, it is not used in clinical anesthesia because the equipment is bulky and heavy, and also it cannot be used to achieve supramaximal stimulus or TOF stimulation.

Q. What are the principles of peripheral nerve stimulation?

Ans. Peripheral nerve stimulator is used for neuromuscular monitoring. A suitable peripheral nerve is stimulated by applying electric current via surface or needle electrodes. The evoked muscle response is either palpated or recorded. The current is usually applied transcutaneously by using ECG (electrocardiogram) electrodes.

A nerve will contain many motor nerve fibers. The reaction of a single muscle fiber to a stimulus follows an all-or-none pattern. In contrast, the response of the whole muscle depends on the number of muscle fibers activated. All of these fibers will need to be stimulated in order to produce a maximal muscle contraction. An electric current of sufficient magnitude would have to be applied for sufficient duration in order to generate an action potential in all of the nerve fibers in a motor nerve. Hence, the nerve has to be stimulated with supramaximal stimulus. Maximal stimulus is the current which generates a response in all nerve fibers and results in maximal muscle contraction. Supramaximal stimulus is a current which is 25% above the maximal stimulus. However, a submaximal current should be used during recovery as supramaximal stimulus can be painful in an awake patient. After administering a neuromuscular blocking agent, the response of the muscle will decrease in parallel to the number of fibers blocked. Therefore, the

reduction in response during constant stimulation reflects the degree of neuromuscular blockade.

The shape of each stimulus should be monophasic and rectangular (also known as square wave stimulus). A biphasic stimulus will produce two responses to a single stimulus due to submaximal stimulation of the nerve by the second part of the biphasic stimulus pulse. The recorded response is a summated response, not suitable for scientific analysis.

The duration of the square wave stimulus should be 0.2–0.3 ms so that it falls within the absolute refractory period of the motor units in the nerve. Pulse duration <0.1 ms will require very high intensity of stimulation because the stimulus duration falls below the rheobase of the motor units. A pulse exceeding 0.5 ms may stimulate the muscle directly or cause repetitive firing.

Q. What are the properties of an ideal nerve stimulator?

Ans. It should be able to generate monophasic square wave electrical stimuli of 0.2–0.3 ms duration of sufficient intensity in the required pattern. The nerve stimulator should be capable of stimulating the nerve at a constant current as compared to a constant voltage, as the current is the determinant of nerve stimulation. It should be able to generate a current of at least 60–70 mA, which is a commonly used range. There should be a digital display of the delivered current. It should be battery operated and small in size. There should be audible signal on delivery of the stimulus. It should have an audible alarm for poor electrode contact. The polarity of the electrode leads should be indicated. Also, it should be capable of delivering the following modes of stimulation: single twitch, TOF, DBS, and PTC. It should have means of recording the evoked response.

Q. What is the current required for supramaximal stimulation?

Ans. The current for supramaximal stimulation is 5–8 mA for needle electrodes and for skin electrodes, it is 20–60 mA depending on the skin resistance (0–2.5 k Ω). A higher current is needed for surface electrodes because the skin resistance has to be overcome to ensure constant stimulating current.

Q. What are the factors which will influence the delivery of constant current?

Ans. Any factor which affects the skin resistance will influence the delivery of constant current. The skin resistance varies with temperature, adequacy of electrode placement, and disease states. Diseased states such as diabetics and chronic renal failure (CRF), and patients with thick and dry skin or obese patients require higher current intensity. Also, cooling the skin would increase the skin

resistance. This reduces the current intensity leading to a decrease in response to stimulation. It is recommended that the negative electrode should be the stimulating electrode. It should be placed directly over the most superficial part of the nerve. The positive electrode should be placed proximally along the course of the nerve, to avoid direct muscle stimulation. ECG electrodes of the silver/silver chloride variety ensure good electrical contact with the skin. The actual conducting area should be small, approximately 7–8 mm in diameter. Otherwise the current produced in the underlying nerve may not be adequate. The skin should always be cleansed adequately (preferably with alcohol swabs) before applying the electrodes. This ensures that there is minimal resistance to the delivery of the current. If supramaximal stimulus cannot be obtained with surface electrodes then needle electrodes can be used.

Q. What are the conditions where neuromuscular monitoring is essential?

Ans.

- When either prolonged infusions of neuromuscular blocking drugs or long-acting neuromuscular blockers are used as in intensive care unit (ICU).
- Prolonged duration of anesthesia using muscle relaxants.
- In certain conditions such as respiratory disease or morbid obesity where inadequate reversal may have devastating effects.
- In conditions where administration of a reversal agent may cause harm, for example, tachyarrhythmias, cardiac failure, and reversal agent are better avoided.
- When pharmacokinetics of muscular relaxants may be altered like in liver or renal dysfunction.
- If muscle relaxants are being used in patients with neuromuscular disorders such as myasthenia gravis and Eaton–Lambert syndrome.
- Surgeries requiring profound neuromuscular blockade, e.g., neurosurgery and vascular surgery in vital areas such as thoracic cavity.

Q. What are the various methods available for quantifying the degree of neuromuscular blockade?

Ans. Visual and tactile assessment of the evoked muscle response cannot be relied upon to assess the degree of neuromuscular blockade. Objective methods of neuromuscular monitoring in which the evoked muscle response is measured and recorded is much more sensitive in excluding clinically significant residual neuromuscular blockade. Five methods are available namely:

1. *Mechanomyography (MMG)*: Measurement of evoked mechanical response of muscle
2. *Electromyography (EMG)*: Measurement of evoked electrical response of muscle

3. *Acceleromyography*: Measurement of acceleration of muscle response
4. *Piezoelectric neuromuscular monitors*: Measurement of evoked electrical response in a piezoelectric film sensor attached to the muscle
5. *Phonomyography*.

Q. What is the principle of MMG?

Ans. Mechanomyography measures evoked muscle tension. The adductor pollicis muscle in the thumb is most commonly used. A strain gauge is attached to the thumb or any other part of the body. The movement of the thumb is limited by a fixed amount of tension (preload). When the ulnar nerve is stimulated at the wrist, the adductor pollicis contracts and causes the thumb to move. However, as the thumb is stabilized, the strain gauge transducer detects a change in tension in the adductor pollicis muscle. This change in tension is measured and converted to an electrical signal, which is then displayed and recorded. The arm and hand must be fixed and the movement of the thumb must be along the length of the transducer to ensure that the readings are accurate. This technique is the gold standard as it can be used for assessment of any pattern of nerve stimulation. However, as it is very cumbersome, it is not routinely used in clinical practice.

Q. What is the principle of EMG?

Ans. Electromyography records the compound action potential that occurs during muscular contraction. Adductor pollicis and ulnar nerve are the most commonly used sites. Five electrodes are used to measure the electrical changes that occur in muscle during stimulation. The two stimulating electrodes are placed over the ulnar nerve. Of the remaining three electrodes, one should be placed over the muscle belly and the reference electrode should be placed over the tendon insertion site. The third electrode is the ground electrode which should be placed between the stimulating and recording electrodes. EMG equipment is easy to assemble and not very bulky. The arm and hand need not be fixed during the recording. Muscles such as diaphragm and larynx can also be monitored, which is not possible with MMG. Motor block during regional anesthesia can be assessed with EMG. Thus, EMG has many advantages over the MMG. However, it also has some disadvantages. Direct muscle stimulation can also cause inaccurate reading and there can be interference from diathermy. Any change in the skin temperature and hand movements can adversely affect the readings to a greater degree than with MMG.

Q. What is the principle of acceleromyography?

Ans. Acceleromyography is measurement of acceleration of the contracting muscle. It is based on Newton's second law—Force = Mass × Acceleration. Acceleration would be

directly proportional to the force if the mass is constant. A piezoelectric transducer is attached to the thumb. When the nerve is stimulated, the movement of the thumb will produce a voltage, which is proportional to its acceleration. This voltage is converted to an electrical signal and displayed. Unrestricted movement of the thumb is required for accuracy. Acceleromyography is mainly used to monitor the TOF ratio.

Q. Are you aware of any other method of measuring the evoked responses?

Ans. Three new methods are available for measuring the evoked responses which are as follows:

1. *Piezoelectric neuromuscular monitors*: They measure the evoked electrical response in a piezoelectric film sensor attached to the muscle. It is based on the principle that stretching or bending a flexible piezoelectric film, in response to nerve stimulation, generates a voltage that is proportional to the amount of stretching and bending.
2. *Phonomyography*: It is a relatively new method. Special microphones are used to record the intrinsic low frequency sounds generated by the contraction of the skeletal muscles. The advantage of this method is that it can be applied to any muscle (diaphragm, larynx, adductor pollicis, and eye muscles) and is easy to apply.
3. *Kinemyography*: There is a piezoelectric sensor placed between the thumb and forefinger. Movement of the thumb following nerve stimulation changes the shape of the sensor material. This results in a flow of current, which is measured as a change in potential that is proportional to the amount of distortion. This technology is used to measure single twitch, TOF, and double burst.

Q. Which is the most common site for neuromuscular monitoring and why?

Ans. Ulnar nerve is the most common site for stimulation. Direct muscle stimulation is avoided as muscle and nerve lie on the opposite side of the arm. The electrodes are placed along the ulnar border of the forearm, with assessment of thumb adduction. The distal (stimulating) electrode should be placed 1 cm proximal to the point at which the proximal flexion crease of the wrist crosses the radial side of the tendon to flexor carpi ulnaris muscle. The proximal electrode should be placed 2–6 cm proximal to the distal electrode (**Fig. 1**). Muscle contraction is easily seen, felt, and quantified and is easily accessible at all times. The adductor pollicis muscle is sensitive to neuromuscular blocking drugs and closely reflects the muscles of the upper airway.

Q. Which other sites can be used for neuromuscular monitoring?

Ans. *Tibial nerve*: Electrodes are placed behind the medial malleolus, with assessment of big toe plantar flexion (**Fig. 1**).

Facial nerve: The stimulating (negative) electrode is placed anterior to the tragus of the ear, with assessment of facial muscle contraction. The positive electrode can be placed over the forehead. Underestimation of the degree of blockade is common, because of direct muscle stimulation and relative insensitivity of the facial muscles to neuromuscular blocking drugs (**Fig. 1**).

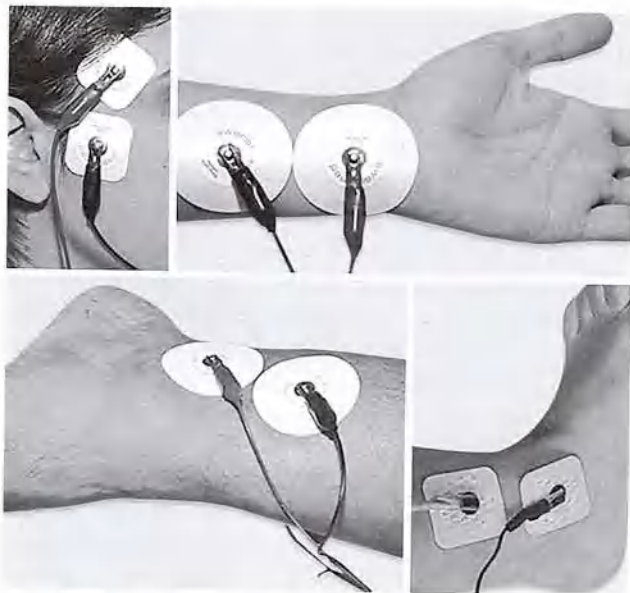


Fig. 1: Placement of negative and positive electrodes in stimulating facial and ulnar nerves and posterior tibial nerve.

Mandibular nerve: Stimulating electrode is placed anterior and inferior to zygomatic arch and the positive electrode is placed over the forehead. Stimulation of the nerve causes closure of the jaw due to contraction of the masseter.

Accessory nerve: One electrode is placed behind the mastoid process and the other at the posterior border of sternomastoid. Stimulation causes the contraction of the sternomastoid and trapezius muscles and is easier to visualize than following stimulation of the facial nerve. There have been reports of asystole following tetanic stimulation when the upper electrode was placed anterior to the ear. It has been attributed to the stimulation of the vagus via the cranial root of the accessory nerve.

Common peroneal nerve: Electrodes are placed lateral to the neck of the fibula, with assessment of foot dorsiflexion.

Q. What are the various patterns of stimulation? Can you describe any one pattern of stimulation?

Ans. Single twitch (0.1–1.0 Hz): This pattern of stimulation is useful before induction to determine the level at which supramaximal stimulus is obtained. A single twitch at 1 Hz (1 twitch every second) is used. The current at which there is no further increase in the evoked response will be the

maximal stimulus. The supramaximal stimulus will be 25% above the maximal stimulus.

A single twitch at 0.1 Hz (1 twitch every 10 seconds) is useful to determine the onset of neuromuscular block. There will be progressively diminished response if the stimulus frequency is >1 Hz. This can result in overestimation of the block. Prerelaxant control response is noted and then subsequent responses are expressed as the percentage of control ($T_1\%$ or T_1/T_c). In both depolarizing and nondepolarizing blocks, there is a progressive decrease in twitch height. The twitch response will only be depressed when a neuromuscular blocking agent occupies 75% of the postsynaptic nicotinic receptors. To assess deep neuromuscular block, single twitches are also used in the PTC, but in this instance, a control twitch height is not required. The major limitation to this technique is the need to measure a control twitch before administering the neuromuscular blocking agent. Also, it cannot distinguish between depolarizing and nondepolarizing block.

Tetanic stimulation (50–100 Hz) for 3–5 seconds may be repeated every 5–10 minutes. It should not be repeated more frequently than every 2 minutes. In a normal muscle, sustained contraction is seen while with a nondepolarizing block, there is a fade in the response. Muscular fatigue may develop at higher frequencies (100–200 Hz) of stimulation. A stimulation frequency of 50 Hz does not cause muscle fatigue and the degree of fade corresponds more closely to the degree of neuromuscular block. Tetanic stimulation does not cause a fade during partial depolarizing block. The amplitude of the evoked response will be lower but the tetanic contraction will be maintained. This pattern of stimulation is very sensitive and can elicit minor degrees of neuromuscular block, which is potentially useful in the postoperative recovery room. However, its use is limited by the fact that it is extremely painful. Also, during the late phase of neuromuscular recovery, tetanic stimulations can cause antagonism of neuromuscular blockade in the stimulated muscle. As a result of this, the response in the tested site may no longer be representative of other muscle groups.

Post-tetanic count: It is used to assess intense neuromuscular blockade. There may be no response to TOF or single twitch stimulation during profound nondepolarizing neuromuscular block. In such situations, a PTC may be useful. A 5-second tetanic stimulus at 50 Hz is administered, followed 3 seconds later by further single twitches at 1 Hz. There would be no response to the single twitches during intense neuromuscular blockade. However, when the blockade dissipates, there would be a first response to post-tetanic twitch stimulation even before the first response to TOF reappears. For any neuromuscular blocking drug, the

number of post-tetanic twitch responses at a given time would be related to the time until the return of the first response of TOF stimulation. It should not be performed more than once in 6 minutes.

Train of four: Four pulses at 2 Hz for 2 seconds, i.e., four stimuli at 0.5-second interval. In depolarizing block, there is equal depression in the twitch height and in nondepolarizing block, there is a fade in the twitch height. Fade in the TOF response after the administration of succinylcholine indicates the development of phase II block. During nondepolarizing block, the TOF count (number of twitches) correlates with the degree of blockade. There is successive decrease in the four responses, with eventual disappearance of the 4th, 3rd, 2nd, and 1st twitches at 75, 80, 90, and 100% blockade, respectively. During recovery, the twitches reappear in the reverse order. The advantages of TOF over other patterns of stimulation are that no control twitch is needed; it is less painful than tetanic stimulation and it does not affect the degree of neuromuscular block.

Double-burst stimulation: DBS was developed to enable the anesthetist to detect even small degrees of neuromuscular block clinically. It is used to assess recovery from nondepolarizing blockade. In DBS, two short bursts of tetanus at 50 Hz at a supramaximal current are applied to a nerve. Typically, each burst will have three impulses (DBS3,3) lasting 0.2 ms. Each impulse is delivered every 20 ms and the two bursts are separated by 750 ms. In unparalyzed muscle, two separate muscle contractions of equal intensity will occur. In muscle partially paralyzed with a nondepolarizing agent, the response to the second burst is reduced. This is the phenomenon of fade. The ratio of the magnitude of the second stimulus to the first is known as the DBS ratio. It is more sensitive at detecting fade than TOF. However, one has to remember that the absence of fade in the tactile response to DBS and TOF does not exclude residual neuromuscular blockade. **Figure 2** shows the various patterns of stimulation.

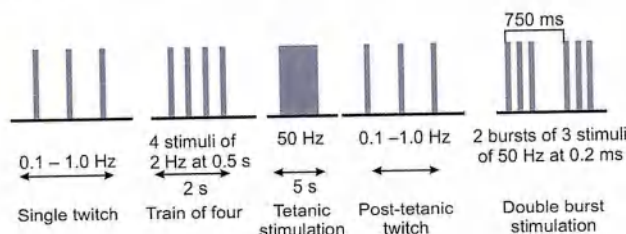


Fig. 2: Various patterns of stimulation.

Q. What do you mean by TOF count and TOF ratio?

Ans. Train-of-four recording demonstrates four phases, or levels, of neuromuscular block: intense block, deep block,

moderate or surgical block, and recovery. When only one response is detectable, the degree of neuromuscular block (the depression in twitch tension) is 90–95%. When the fourth response reappears, neuromuscular block is usually 60–85%.

Train-of-four count is the number of palpable muscle twitches. Twitch suppression of 90% would equate to a TOF count of 1 or less.

Train-of-four ratio is the force of the first twitch divided by the force of the fourth. It may be repeated every 10–15 seconds.

Suggested suitable values during anesthesia are as follows:

- TOF count of 1 or PTC should be zero for tracheal intubation.
- TOF count of 1–2 for maintenance; deeper levels may be required for complete diaphragmatic paralysis.
- TOF count of 3–4 before attempting reversal of blockade, especially with long-acting drugs.

Relationship between TOF ratio and neuromuscular recovery is described as follows:

- TOF ratio 0.4—patient is unable to lift the head or arm. Tidal volume may be normal but vital capacity and inspiratory force may be reduced.
- TOF ratio 0.6—most patients are able to lift the head for 3 seconds, open their eyes widely, and protrude their tongue, but inspiratory force and vital capacity is reduced.
- TOF ratio 0.7—patient can cough and lift their head for at least 5 seconds but their grip strength is low.
- TOF ratio >0.8—vital capacity and inspiratory force are normal, but patient may have diplopia and facial weakness.
- TOF ratio (at the thumb) of 0.9 for adequate maintenance of spontaneous ventilation. Studies have shown that if TOF ratio is <0.9, there is functional impairment of muscles of pharynx and upper esophagus, which can predispose to regurgitation and aspiration.

Q. Why do you get a fade on tetanic stimulation?

Ans. Fade in response to tetanic stimulation is considered a presynaptic event. At the neuromuscular junction, the acetylcholine receptors (AChRs) are of two types, i.e., nicotinic and neuronal. The classic muscle-type nicotinic AChR is postsynaptically present, whereas neuronal subtype receptors may be presynaptically and postsynaptically present. The nicotinic receptor on the junctional surface of the nerve senses transmitter in the cleft and, by means of a positive-feedback system, causes release of more transmitters. When the concentration of transmitter in the synaptic cleft has appropriately increased, by virtue of negative-feedback mechanism, further release of

acetylcholine shuts down. Tetanic stimulation results in the release of large amounts of acetylcholine release from the immediately available stores in the nerve terminal. After administration of nondepolarizing muscle relaxants (NDMRs), tetanic stimulation will result in a fade. This is because the prejunctional receptor is especially blocked by the NDMRs leading to loss of positive-feedback mechanism with subsequent fade phenomenon. Succinylcholine neither activates nor inhibits the presynaptic $\alpha_3\beta_2$ autoreceptor at clinically relevant concentration. Hence, the classical fade phenomenon is not observed with depolarizing agents. Nevertheless, these prejunctional receptors are blocked if much higher concentrations of succinylcholine are administered. This is called phase 2 block.

Q. What is post-tetanic facilitation?

Ans. During moderate nondepolarizing block, if you give tetanic stimulation (50 Hz) for 5 seconds followed by post-tetanic twitch stimulation (1.0 Hz), there is an increase in the twitch height, which is due to post-tetanic facilitation of transmission. This occurs because tetanic stimulation causes mobilization and synthesis of acetylcholine which continues for some time even though the stimulation has stopped. The degree and duration of post-tetanic facilitation depends on the degree of neuromuscular blockade.

Q. What are the clinical signs of postoperative neuromuscular recovery?

Ans. Unreliable—sustained eye opening, protrusion of tongue, arm lift to opposite shoulder, normal tidal volume, normal or near normal vital capacity, and maximum inspiratory pressure <40 – 50 cmH₂O.

Reliable—sustained head lift for 5 seconds, sustained leg lift for 5 seconds, sustained hand grip for 5 seconds, sustained tongue protrusion, maximum inspiratory pressure >40 – 50 cm of H₂O, and effective cough or eye opening for 5 seconds without any diplopia. The ability to resist the removal of a tongue depressor from between the clenched teeth would be a more sensitive test.

Q. Is neuromuscular monitoring reliable in hypothermic patients?

Ans. No, neuromuscular monitoring is not reliable in hypothermic patients. Both central and peripheral cooling reduce the twitch tension and TOF ratio. Cooling affects nerve conduction and reduces the rate of acetylcholine release and muscle contraction. Cooling reduces the current delivery by increasing skin impedance. This results in a submaximal response. Also, cooling causes vasoconstriction, which reduces the blood flow to the muscles which in turn decreases the rate of removal of muscle relaxant from the neuromuscular junction. As a result of all these factors, the evoked muscle response will be different between warm and cold extremity.

Q. What is your choice of stimulation pattern and which muscle would you monitor during the induction of anesthesia?

Ans. The central muscles such as diaphragm and larynx have a very good blood supply, as a result of which the onset and offset of the block is faster. The peripheral muscles such as adductor pollicis have a poor blood supply due to which the onset of block is delayed. However, they are very sensitive to neuromuscular blockers due to which the recovery is slow. The muscles of upper airway and pharynx behave like the central muscle at onset (faster onset) and like the peripheral muscles during recovery (delayed recovery). Also, as compared to the peripheral muscles, the diaphragm is the most resistant to both depolarizing and nondepolarizing agents. During the induction of anesthesia and tracheal intubation, the diaphragm and the muscles of the jaw and larynx must be completely paralyzed. If the adductor pollicis is used to monitor the neuromuscular function during induction, then the absence of response to TOF would not ensure complete paralysis of the diaphragm. The orbicularis oculi is probably the ideal muscle to monitor at the time of induction as it is more similar to a central muscle—onset of block will be similar to the laryngeal muscles and diaphragm.

During induction, single twitch stimulation should be used to determine the supramaximal stimulus. During intubation, TOF count of 1 will correspond to optimal intubating conditions.

Q. Which muscle should be monitored during maintenance of anesthesia and why?

Ans. The diaphragm is relatively resistant to neuromuscular block and it recovers from paralysis more quickly than the peripheral muscles. If you monitor a more sensitive peripheral muscle, such as the adductor pollicis, it may not adequately reflect the degree of block required during the maintenance of anesthesia. A central muscle which is resistant to neuromuscular block, for example, corrugator supercilii muscle, will reflect the diaphragm more closely and should be monitored during the maintenance of anesthesia.

During the maintenance of anesthesia, the TOF count should be 1 or 2. For profound neuromuscular block, the PTC should be zero.

Q. Which muscle should be monitored during reversal?

Ans. During reversal, a peripheral muscle, such as adductor pollicis, should be monitored. As the peripheral muscles have a slower recovery time, the recovery of the peripheral muscles would indicate the respiratory muscles have recovered to a greater degree. This provides a larger margin of safety.

Before administering a neuromuscular antagonist, the TOF count should be at least 3 or the TOF ratio should be 0.7.

Q. When should the reversal be given?

Ans. The neuromuscular blockade is reversed by two mechanisms. The first mechanism is by recovery from the NDMR drug itself which occurs by metabolism and elimination. The second mechanism which hastens this process is by increasing the concentration of acetylcholine at the receptor site which offers more competition to these drugs. This can be done by administering reversal agents which are acetylcholinesterase inhibitors.

Antagonism of neuromuscular block with neostigmine should not be attempted when the block is intense or deep. Even if some reversal occurs, it will often be inadequate, regardless of the dose of neostigmine administered. The ceiling effect of anticholinesterases is an important limitation of reversal agents, neuromuscular blockade cannot be adequately reversed if high concentrations of NMDBs are present at neuromuscular junction. Reversal should not be initiated before at least two to four responses are observed.

Q. If you are using neuromuscular monitoring, then when would you decide to extubate the patient?

Ans. While extubating any patient all four responses to TOF should be present and the strength of all four responses should be the same (TOF ratio of 0.9:1). However, visual and tactile assessment of the strength of the stimulus is not very sensitive. The strength of DBS is better appreciated than the stimulus strength of TOF. Hence, use DBS to assess the adequacy of reversal if you do not have a monitor which can measure the TOF ratio. However, it is important to remember that tactile evaluation is not a very sensitive

method and even though both responses to DBS appear same in magnitude, it still does not exclude clinically significant residual blockade. Hence, manual evaluation of responses to nerve stimulation should always be considered in relation to reliable clinical signs and symptoms of residual neuromuscular blockade. Objective assessment is ideal and to exclude clinically significant residual neuromuscular blockade, the TOF ratio should be >0.9 .

Q. What are the recent applications of PNS?

Ans. The peripheral nerve stimulation can be used to treat pain. This application is based on the gate theory. The gate theory proposed that activation of large diameter afferent fibers have an inhibitory effect on the central transmission of small diameter afferent fibers, thereby blocking pain perception. The options for electrical nerve stimulation are via the transcutaneous or directly implantable routes. It is being used in certain complex regional pain syndromes, pain due to peripheral nerve injuries, treatment of back pain, occipital nerve stimulation for the treatment of migraine headaches, and pudendal nerve stimulation that is being investigated for use in urinary bladder incontinence.

■ SUGGESTED READING

1. Dalrymple P, Chelliah S. Electrical nerve locator. Continuing Education in Anaesthesia, Critical Care and Pain. 2006;6(1):32-6.
2. Dorsch JA, Dorsch SE. Understanding Anesthesia Equipment, 5th edition. Philadelphia: Lippincott Williams and Wilkins; 2007.
3. Kalra P. In: Miller RD (Ed). Millers Anesthesia, 7th edition. Philadelphia: Churchill Livingstone; 2009.
4. Mcgrath CD, Hunter JM. Monitoring of neuromuscular block. Continuing Education in Anaesthesia, Critical Care and Pain. 2006;6(1):7-12.

Oxygen Therapy Devices and Manual Resuscitator

Nayana Amin, Vijaya Prakash Patil, Sohan Lal Solanki

Q. Which patients need oxygen therapy?

Ans. American College of Chest Physicians and National Heart, Lung, and Blood Institute recommendations for instituting oxygen therapy are:

- Cardiac and respiratory arrest
- Hypoxemia ($\text{PaO}_2 < 60$ mm Hg, $\text{SaO}_2 < 90\%$)
- Hypotension (systolic BP < 100 mm Hg)
- Low cardiac output and metabolic acidosis
- Respiratory distress (respiratory rate > 24 breaths/min).

While providing oxygen therapy, attempts must be made to find the underlying pathology and treat it.

Q. What is the difference between hypoxia and hypoxemia?

Ans. Hypoxia is lack of oxygen at the tissue level whereas hypoxemia is low oxygen tension (< 60 mm Hg) in the arterial blood.

Q. What are the possible causes of hypoxia?

Ans. Hypoxia could be due to arterial hypoxemia or failure of the oxygen hemoglobin transport system.

Arterial hypoxemia:

- Low inspired oxygen partial pressure (high altitude)
- Alveolar hypoventilation (sleep apnea, narcotic overdose, neuromuscular disorders, anesthetics, and sedatives)
- Ventilation-perfusion mismatch (atelectasis, pneumonia, acute asthma)
- Right to left shunts (pneumonia, pulmonary embolism, arteriovenous malformation)
- Failure of oxygen hemoglobin transport system
- Inadequate tissue perfusion (cardiac failure, myocardial infarction, hypovolemic shock)
- Low hemoglobin concentration (anemia, trauma)
- Abnormal oxygen dissociation curve (hemoglobinopathies, high carboxyhemoglobin)

- Increased oxygen requirements in hypermetabolic states
- Histotoxic poisoning of intracellular enzymes (cyanide poisoning, septicemia).

Depending on the cause and severity, oxygen can be supplemented from a range of oxygen therapy devices. These devices deliver oxygen at or just above atmospheric pressure (normobaric oxygen therapy). There are some patients in whom, either the oxygen carrying capacity of hemoglobin is compromised (e.g., carbon monoxide poisoning) or extra tissue oxygen is required (in severe burns and tissue infections). In these patients, supplemental oxygen may be delivered by dissolving it under pressure, in plasma (hyperbaric oxygen therapy).

Q. What are the indications for oxygen therapy in the postoperative period?

Ans. The indications for oxygen therapy would be:

- *Patient factors:* Cardiorespiratory disease, elderly, shivering, obesity.
- *Surgical factors:* Thoracic surgery, upper abdominal surgery.
- *Physiological factors:* Hypovolemia, hypotension, anemia.
- *Postoperative analgesic technique:* Patient controlled analgesia, IV opioid infusion.

Q. How would you classify oxygen therapy devices?

Ans. There are a number of methods of classifying oxygen therapy devices. They can be classified as follows:

- *Performance of the device:* Fixed (Venturi mask) or variable performance (nasal prong, face mask).
- *Flows delivered by the device:* Low flow (nasal prongs, face mask) or high flow (Venturi mask).
- *Patient:* Dependent [face mask, nasal prongs, continuous positive airway pressure (CPAP) device] or patient-independent (ventilators).

- *Degree of dependency:* Low dependency (face mask, nasal prongs), medium dependency (CPAP mask and equipment), high dependency (noninvasive or invasive positive pressure ventilation).

Q. What is the importance of peak inspiratory flow rates while choosing the oxygen therapy devices?

Ans. If a patient is breathing spontaneously from a T-piece with no reservoir and zero fresh gas flow (FGF), then all the inspired gas would be room air. If the FGF is increased to 100 L/min, then all the inspired gas would be taken from the FGF and the FiO_2 will be the same as the fresh gas. If the FGF are decreased from 100 L/min, there will be a point at which the FGF will not meet the patient's requirements. Below this flow, the patient will entrain room air which will dilute the FiO_2 of the fresh gas. This occurs when the FGF is less than the peak inspiratory flow.

For example: If a patient is breathing at a rate of 10 breaths/min, tidal volume is 500 mL and inspiratory-to-expiratory ratio is 1:2 then each respiratory cycle will be of 6 seconds with an inspiratory time of 2 seconds. To inspire a tidal volume of 500 mL over 2 seconds, the mean inspiratory flow should be 0.25 L/s or 15 L/min. If the patient is breathing with a square wave pattern, the peak inspiratory flow would be the same as the mean inspiratory flow. However, if the breathing pattern is sine wave the peak flow must be higher than the mean flow. Normally, the peak inspiratory flow rates are 60–70 L/min and it can be higher in patients who are hyperventilating or exercising.

If a patient receives a FGF of 10 L/min then at the start of inspiration, all inspiratory gas will be taken from FGF. However, entrainment of room air will occur when the inspiratory flow exceeds 10 L/min. After this point, the FiO_2 will depend on the amount of room air that is entrained. Therefore, in order to deliver a known and fixed FiO_2 to the patient via a face mask, the FGF should exceed peak inspiratory flow.

Q. What do you mean by fixed or variable performance devices? Give examples of each.

Ans. Supplemental oxygen can be supplied by a variety of devices. The effectiveness of each device is determined by the capacity of each device to deliver sufficient oxygen at high enough flow rate to match the patients spontaneous inspiratory flow rate. In a nonintubated

patient breathing in an "open" system the capacity of the oxygen therapy device to meet the patient's inspiratory flow will determines how much room air will be entrained. The FiO_2 delivered from the oxygen source will be diluted by the entrained room air. Therefore, the oxygen delivery systems are categorized as either variable performance (no control on FiO_2) or high-flow fixed performance (controlled FiO_2) systems.

The variable performance systems are "patient-dependent" because the FiO_2 that the patient receives will change with changes in the respiratory parameters. For example, nasal catheters, nasal cannulae, and masks with or without a rebreathing bag.

The fixed performance systems are usually considered as "patient-independent" because regardless of changes in respiratory parameters, the patient will receive a constant, predetermined inspired oxygen concentration (FiO_2). For example, Ventimask.

Q. What are the factors which control the FiO_2 in variable performance devices?

Ans. In variable performance devices FiO_2 varies with the phase and pattern of respiration. Entrainment of room air occurs during the use of these devices, as the oxygen flow delivered by these devices is less than patient's peak inspiratory flow. During normal respiration the rate and tidal volume varies with each breath (**Fig. 1**). The exact proportion of air dilution cannot be known due to the irregular respiratory pattern. As a result of this, the FiO_2 not only varies with each breath but also varies from patient to patient.

The factors which control the FiO_2 are:

- *Equipment factors:* Oxygen flow rate, mask volume (100–200 mL) and volume of the reservoir bag (600–1,000 mL), fit of the mask to the face, size and placement of the vents.
- *Patient factors:* Respiratory rate, tidal volume and peak inspiratory flow rates, and anatomical reservoir. The anatomical reservoir varies with the size of the subject. In infants, the anatomical dead space is 3.3 mL/kg. It decreases to the adult value of approximately 2 mL/kg by the age of 6 years. During the period of development, the intrathoracic anatomical dead space remains constant at 1 mL/kg while the volumes of the nose, mouth, and pharynx change relative to the body weight. From

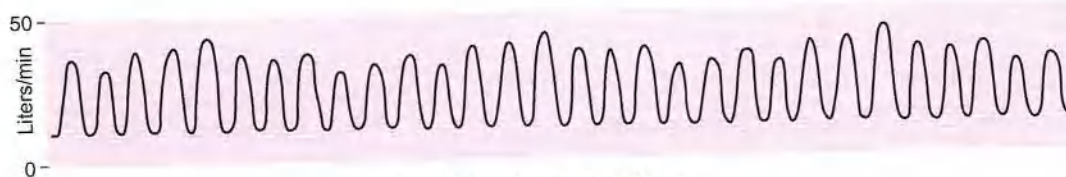


Fig. 1: Variation in tidal volume.

early adulthood, the anatomic dead space increases by approximately 1 mL/year.

- *Additional factors:* Presence of other gases or vapors, e.g., humidifier.

Q. Can you explain how the FiO_2 varies with a variable performance device?

Ans. We have already mentioned the various factors that affect the fraction of inspired oxygen (FiO_2).

When giving oxygen therapy there is pooling of oxygen in the anatomical reservoir of the upper airways, at the end of exhalation. The oxygen collected in the anatomical and the equipment reservoir is available for the next inspiration. The anatomical reservoir (nose and nasopharynx) is 50 mL, mask space varies from 100 to 200 mL, and reservoir bag if present may have a volume of 600–1,000 mL. Depending on the reservoir capacity it may or may not be able to meet the tidal volume requirements. If the oxygen flow rate is constant then the FiO_2 will be low if the tidal volume, respiratory rate and peak inspiratory flow rates are high.

Consider the following example of a normal breathing pattern: Tidal volume (VT) 500 mL, respiratory rate 20 breaths/min, inspiratory time 1 second, expiratory time 2 seconds (including end-expiratory pause 0.5 second), anatomic reservoir 50 mL. If this patient is receiving oxygen at 2 L/min what would the FiO_2 be?

- $2 \text{ L/min} = 33 \text{ mL/s}$ ($2,000 \text{ mL/60 s}$)
- 17 mL of 100% oxygen from the anatomic reservoir ($0.5 \text{ second end-expiratory pause} \times 33 \text{ mL/s}$)
- 33 mL of 100% oxygen during inspiration (inspiratory time 1 second)
- 450 mL of 21% oxygen (room air) = 95 mL of 100% oxygen
- $17 + 33 + 95 = 145 \text{ mL of 100% oxygen}$
- $\text{FiO}_2 = 145 \text{ mL of oxygen/500 mL} = 0.29\%$
- $6 \text{ L/min} = 100 \text{ mL/s}$ ($6,000 \text{ mL/60 s}$)
- 50 mL of 100% oxygen from the anatomic reservoir ($0.5 \text{ second end-expiratory pause} \times 100 \text{ mL/s}$) 100 mL of 100% oxygen during inspiration (inspiratory time 1 second)
- 350 mL of 21% oxygen (room air) = 73 mL of 100% oxygen
- $50 + 100 + 73 = 223 \text{ mL of 100% oxygen}$
- $\text{FiO}_2 = 223 \text{ mL of oxygen/500 mL} = 0.44\%$
- If VT = 500 mL and RR is 10 then inspiratory time is 2 seconds, expiratory time 4 seconds, end-expiratory pause 0.5 second. Now doing the above calculations with $2 \text{ L/min} = 33 \text{ mL/s}$ ($2,000 \text{ mL/60 s}$)
- 17 mL of 100% oxygen from the anatomic reservoir ($0.5 \text{ second end-expiratory pause} \times 33 \text{ mL/s}$)
- 66 mL of 100% oxygen during inspiration (inspiratory time 2 seconds $\times 33 \text{ mL}$)

- 417 mL of 21% oxygen (room air) = 87 mL of 100% oxygen
- $17 + 66 + 87 = 170 \text{ mL of 100% oxygen}$
- $\text{FiO}_2 = 170 \text{ mL of oxygen/500 mL} = 0.34\%$.

These are just approximate values. There are various other factors which can influence the FiO_2 like changes in the tidal volume or the end-expiratory pause.

Q. What do you mean by low-flow system? Give examples.

Ans. Oxygen therapy devices are also classified as high- or low-flow systems, depending on the total gas flows that are delivered to the patient. Low-flow systems deliver oxygen at flows that are less than the patient's inspiratory flow rate leading to air entrainment. As a result of this inhaled oxygen concentration (FiO_2) may be low or high, depending on the specific device and the patient's inspiratory flow rate. As oxygen delivery of low-flow systems varies with the patient's inspiratory flow they are also classified as variable performance oxygen delivery systems. However, an anesthetic breathing system which has a tight-fitting mask and a reservoir is a low-flow system which can deliver a known FiO_2 . Examples of low-flow systems:

- *No capacity system:* Nasal cannula, nasopharyngeal catheters.
- *Low capacity system (capacity <100 mL):* Simple and nebulizer face mask for children, tracheostomy mask.
- *Medium capacity system (capacity 100–250 mL):* Simple and nebulizer face mask for adults.
- *High capacity system (capacity 250–1,500 mL):* Face mask with reservoir bag.
- *Very high capacity system (capacity >1,500 mL):* Incubators, oxygen hood, oxygen tents.

Q. How would you describe any oxygen therapy device?

Ans. First classify the device according to the flows (low-flow or high-flow device), the performance of the device (variable or fixed performance) and the capacity of the device (no capacity, low capacity, medium capacity or large capacity). Then give a brief description of the device. What is the FiO_2 delivered, where it is used, what are the advantages, disadvantages, and limitations of the device? How is the device sterilized?

Q. Can you give a brief description of any low-flow oxygen delivery system?

Ans.

No Capacity Systems

Nasal cannulae: These are low-flow variable performance devices with no capacity. They consist of two soft prongs that arise from oxygen supply tubing. The prongs are inserted into the patient's anterior nares, and the tubing is secured to the patient's face by an adjustable strap. Oxygen is delivered through the cannula, but at low-flow rates between 0.5 and

5.0 L/min. The patient's nasopharynx acts as an anatomic reservoir. When the patient inspires, the oxygen is diluted with room air. The degree of dilution depends on the patient's inspiratory flows. The resulting FiO_2 depends on the flow rate of oxygen, patient's minute ventilation and inspiratory flow and the volume of the nasopharynx. The FiO_2 cannot therefore be precisely controlled, but the maximal tracheal FiO_2 is not likely to exceed 0.4 (40%). Mouth breathing causes inspiratory airflow. This produces a Venturi effect in the posterior pharynx entraining oxygen from the nose.

Approximately, nasal cannulae deliver 4% oxygen per liter:

- Flow 1 L/min: 24%
- Flow 2 L/min: 28%
- Flow 3 L/min: 32%
- Flow 4 L/min: 36%
- Flow 5 L/min: 40%.

Higher flow rates should not be used as they have a drying and irritating effect on the nasal mucosa and do not result in much higher FiO_2 . High flows can cause headaches and nose bleeds. Since it is a variable performance system a fixed FiO_2 cannot be assigned to patients on low-flow system. Patients are more compliant with the nasal cannula than the face mask as they are able to speak, eat, and drink (**Fig. 2**).

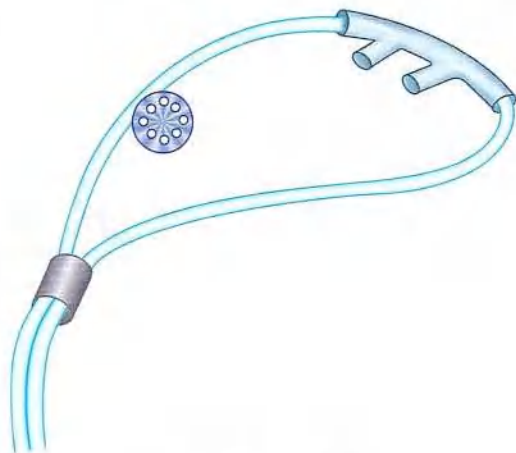


Fig. 2: Nasal cannula.

Advantages of nasal cannula:

- Comfortable for patient
- Ideal for claustrophobic patients
- Ideal for oxygen-dependent patients requiring small amounts of oxygen especially at home
- Humidification not required up to 4 L/min.

Disadvantages of nasal cannula:

- Unpredictable FiO_2
- Maximum estimated FiO_2 0.4
- Not appropriate for patients in respiratory distress.

Limitations:

- Any change in the patient's respiratory pattern can cause air entrainment leading to fluctuations in FiO_2
- Prongs are difficult to keep in position, especially in small infants
- Use may be limited by the presence of excessive mucus drainage, mucosal edema, or a deviated septum
- Maximum flow should not be >2 L/min in infants and newborns.

Equipment-related potential harm:

- Skin irritation can result from material used to secure the cannula or from local allergic reaction to polyvinyl chloride
- Improper sizing can lead to nasal obstruction or irritation
- Displacement can lead to loss of oxygen delivery
- Inadvertent CPAP may be administered depending upon the size of the nasal cannula, the gas flow, and the infant's anatomy
- Irritation and drying of nasal mucosa can result if flows are excessive.

Nasopharyngeal catheters: They are variable performance low-flow device with no capacity. They are soft tubes with several distal holes. Catheters are available from 8 to 14 Fr size. The catheter should be well-lubricated prior to insertion. The depth of insertion should be equal to the distance from the ala nasi to the tragus. The tube is secured to the patient's face and connected to oxygen supply tubing. Oxygen flows from the catheter into the patient's oropharynx, which acts as an anatomic reservoir. The FiO_2 varies with the patient's inspiratory flow. The patient should be observed for evidence of catheter occlusion, and the catheter should be alternated between nares every 8–12 hours and changed daily.

Limitations:

- Method is in less common use because of the complexity of care.
- FiO_2 is difficult to control and measure.
- Its use may be limited by excessive mucus drainage, mucosal edema, or the presence of a deviated septum.
- Catheter should be cleared frequently to prevent occlusion of the distal holes.
- Catheter sizes <8 Fr are less effective in oxygen delivery.
- Lower oxygen concentrations are delivered if the catheter is placed in the nose rather than in the pharynx.

Equipment-related potential harm:

- Improper insertion can cause gagging and nasal or pharyngeal trauma
- Improper sizing can lead to nasal obstruction or irritation

- Excessive oxygen flow can produce pain in the frontal sinuses
- Excessive secretions and/or mucosal inflammation can result
- Skin irritation may result from material used to secure the cannula and/or from local allergic reaction to polyvinyl chloride
- Occlusion of distal openings may occur
- Excessive flow may cause gastric distention.

Small Capacity Systems

Simple oxygen masks: These are low-flow variable performance device with small capacity. The pediatric face masks have low capacity (70–100 mL) and the adult facemask have medium capacity (100–250 mL). They are plastic reservoirs designed to fit over the patient's nose and mouth. An elastic strap is used to secure the mask around the patient's head. Higher FiO_2 can be delivered if the mask volume is increased. Some masks may have a deformable metal which allow the mask to be shaped around the nose to achieve a closer fit. Oxygen is delivered through a small-bore tube connected to the base of the mask. The oxygen that gets collected in the apparatus dead space at the end of expiration is inhaled at the beginning of the next breath. If there is no expiratory pause, alveolar gas may be rebreathed from the mask at the start of inspiration. Exhaled gases are vented out through the holes on each side of the mask, which also serve as room-air entrainment ports. As it is a low-flow device there is air entrainment during inspiration and the FiO_2 varies with the patient's inspiratory flow, mask fit, and patient respiratory pattern. The FiO_2 can be reduced if the spontaneous inspiratory flows are high, mask is loose or oxygen flow is low.

Simple oxygen masks can provide 35–60% FiO_2 , depending on fit, at flow rates from 5–10 L/min (Table 1). Caution should be taken when using a simple mask where accurate delivery of low concentrations of oxygen is required (Fig. 3).

TABLE 1: Flow rate and FiO_2 with simple mask.

O_2 flow rate (L/min)	FiO_2
5–6	0.4
6–7	0.5
7–8	0.6

Advantages of simple oxygen mask: Quick and easy to set-up and apply.

Disadvantages of simple oxygen mask:

- Unpredictable FiO_2
- Not >0.5 FiO_2 can be delivered.

Watch for: CO_2 rebreathing can occur with flow rates <2 L/min or if minute ventilation is very high.

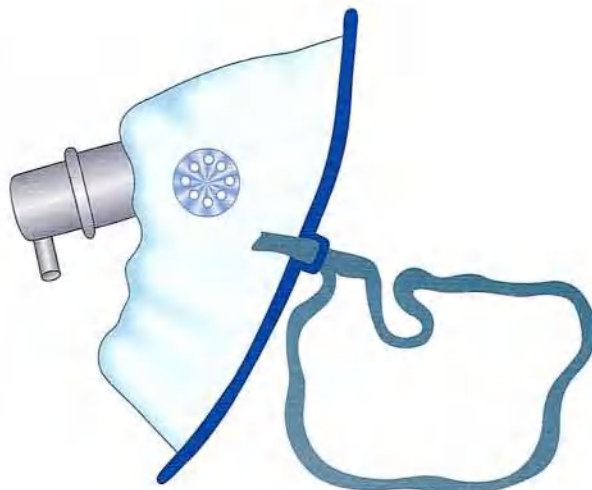


Fig. 3: Simple oxygen mask.

Aerosol (nebulizer) face mask: It is a variable-oxygen, low-flow device with medium capacity. The mask with large side holes is attached by large-bore tubing to a nebulizer, which delivers water/drug in aerosol form. Humidified oxygen can be delivered with a nebulizer mask.

Advantages of nebulizer face mask: Humidified gases.

Disadvantages of nebulizer face mask:

- Nebulizer is noisy
- Unpredictable FiO_2
- Maximum FiO_2 estimated at 0.50 (same as plain mask).

Watch for: Attempts to minimize noise by decreasing oxygen flow on flowmeter will interfere with effective nebulization and thus humidification.

Tracheostomy mask: Tracheostomy mask is low-flow variable performance device with low capacity.

Advantages of tracheostomy mask: It is designed for patients with a tracheostomy.

Disadvantages of tracheostomy mask: It can require frequent cleaning or replacement if patient is coughing up sputum.

High Capacity Systems

In all of the above systems, most of the oxygen stored in device during exhalation gets wasted as it is blown away by the patient's exhaled gases. Oxygen also gets wasted if the oxygen flow rates are more than the patient's inspiratory flow rates. The large capacity devices have been designed to store this wasted oxygen.

Reservoir face mask: It is a high-oxygen, low-flow, variable performance device. These masks provide a higher FiO_2 as there is a reservoir bag attached to the face mask. Oxygen flows directly into the reservoir bag. The flow rate of oxygen is adjusted so that the bag remains completely or partially distended throughout the respiratory cycle. The patient

inhales oxygen preferentially from the reservoir. If the reservoir empties during inspiration, then ambient air is entrained from the vents in each side of the mask body if necessary. Oxygen delivery can be maximized by using a mask with a good fit. FiO_2 varies depending on mask fit, capacity of reservoir bag, oxygen flow rates, and patients' inspiratory flow.

The reservoir face mask can be a partial rebreathing mask or a nonrebreathing mask (**Fig. 4**).

Partial rebreathing mask: It is a simple mask with a reservoir bag. Oxygen flow should always be supplied to maintain the reservoir bag at least one-third to one-half full on inspiration. The reservoir receives fresh gas plus exhaled gas approximately equal to the volume of the patient's anatomic dead space. The oxygen concentration of the exhaled gases combined with the supply of fresh oxygen, permits the use of flows lower than those necessary for other devices (e.g., nonrebreathing masks), and potentially conserves oxygen use. At a flow of 6–10 L/min the system can provide 40–70% oxygen.

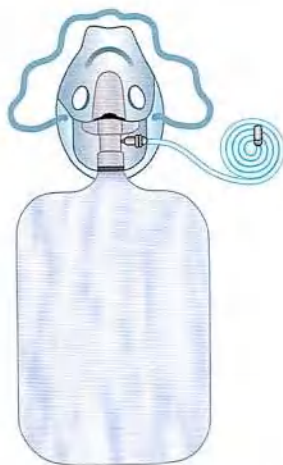


Fig. 4: Reservoir face mask.

Nonrebreathing masks: They are similar to partial rebreathing masks but do not permit the mixing of exhaled gases with the fresh gas supply. A series of one-way valves ensures a fresh oxygen supply with minimal dilution from the entrainment of room air. The one-way valve over the reservoir bag prevents entry of expired gas, and the one-way valve over one of the side ports limits entrainment of room air during inspiration. As the flap valves are on the outside they do not impede exhalation. This design provides a higher FiO_2 than the simple and partial-rebreathing masks and the nasal devices provided the mask fits correctly. There should be a minimum flow of 10 L/min. The delivered FiO_2 of this system is 75–90% at flow rates of 12–15 L/min. Some of these masks may have a device which gives a visual

indication of the patient's respiratory efforts (respiratory-check face mask) (**Table 2**).

TABLE 2: Flow rate of FiO_2 with reservoir face mask.

O_2 flow rate (L/min)	No valves	One-way valve
3	0.5–0.65	0.45–0.6
6	0.6–0.75	0.6–0.75
9	0.65–0.80	0.65–0.80
12	0.7–0.85	0.7–0.85
15	0.75–0.9	0.75–0.9

High inspired oxygen concentrations can be achieved with:

- High oxygen flow rates
- Large reservoir bags
- Inclusion of flap valves
- Slower respiratory rates (to allow reservoir bags to fill)
- Smaller tidal volumes.

Advantages of reservoir face mask: Fast and easy to set-up.

Disadvantages of reservoir face mask: FiO_2 is variable if there is air entrainment due to poor mask fit.

Watch for:

- Reservoir bag must always remain inflated. Deflated bag indicates that the oxygen flow rates are low.
- These masks cannot be used for long-term as there is no humidification of gases.

Limitations:

- FiO_2 may be unpredictable depending on inspiratory flow and construction of the mask's reservoir.
- May not be well-tolerated by claustrophobic patients.
- May interfere with feeding.
- The maximum FiO_2 attainable with a simple, nonrebreathing or partial rebreathing mask in neonates, infants, and children has not been well-documented.
- Performance is altered if the entrainment ports are blocked.

Equipment-related potential harm:

- Aspiration of vomitus may be more likely when a mask is in place in patients with obtunded airway reflexes.
- Irritation may result from tight application.
- Rebreathing of carbon dioxide may occur if total oxygen flow is inadequate.
- Not appropriate for use in neonates.

Q. A 35-year-old male patient, who is very tachypneic, is receiving oxygen therapy via a Hudson mask with oxygen flows of 6 L/min. He is maintaining a saturation of 90%. Reservoir mask is not available. The nurse thinks that she

can increase the FiO_2 if she closes all the leaks around the mask including the holes in the side of the mask by applying sticking plaster. Is she doing the right thing?

Ans. No, she is not doing the right thing. The Hudson mask is a low-flow device. With 6 L/min of oxygen flows approximately 54 L of room air would have to be entrained to match the patient's peak inspiratory flow rates. The air entrainment occurs from the holes at the sides of the mask. If there is a tight seal, there would be no air entrainment and the patient would feel suffocated. Also the holes at the side are for the egress of exhaled gases. Closure of these holes would result in rebreathing.

Q. What do you mean by a high-flow system? Give example.

Ans. High-flow systems are fixed-performance oxygen delivery systems as they can provide a specific FiO_2 at flows that meet or exceed the patient's inspiratory flow requirement. While there is no defined cut-off point between low and high flow, most authors place it at about 10–15 L/min. These devices create a constant proportion of air-oxygen mixture in excess of patient inspiratory flow rate and are independent of patient factors or fit to the face. There is no rebreathing with these devices as the CO_2 gets washed out by the gas flow which constantly exceeds the patient's demand. However, when the flow is less than the peak inspiratory flow, even the high flow devices become patient-dependent.

To work efficiently and to prevent air dilution a fixed performance device should incorporate:

- A large volume face piece (not <280 mL)
- Gas mixture flowing directly toward the nose and mouth
- Vents positioned well away from the patient airway. For example, Ventimask.

Q. What is Venturi effect?

Ans. The Venturi effect is based on the Bernoulli principle. The relationship between the velocity and pressure exerted by a moving liquid is described by the Bernoulli's principle: as the velocity of a fluid increases, the pressure exerted by that fluid decreases. This principle can be applied to both liquids and gases.

When a fluid flows through a constriction the fluid velocity must increase to satisfy the equation of continuity. Due to the principle of conservation of energy, any gain in kinetic energy due to the increase in fluid velocity must be balanced by a drop in pressure distal to the constriction.

The Venturi principle describes how a second fluid can be entrained into the stream of the first. It can either be done through a coaxial arrangement or through a side arm opening into an area of low pressure.

Q. Which other devices are based on the Bernoulli principle?

Ans. The Venturi effect may be observed or used in the following:

- Venturi masks used in medical oxygen therapy.
- Sanders jet injector.
- Inspirators that mix air and flammable gas in grills, gas stoves, Bunsen burners.
- Steam siphons use the kinetic energy from the steam pressure to create a partial vacuum.
- Water aspirators that produce a partial vacuum using the kinetic energy from the faucet water pressure.
- Atomizers that are used to disperse perfume or spray paint.
- Foam firefighting nozzles and extinguishers.
- Carburetors use the Venturi effect to suck gasoline into an engine's intake air stream.
- Industrial vacuum cleaners which operate on compressed air.
- Venturi scrubbers used to clean fuel gas emissions.
- Injectors used to add chlorine gas to water treatment chlorination systems.
- Steam injectors use the Venturi effect to deliver feed water to a steam locomotive boiler.
- Sand blasters used to draw fine sand in and mix it with air.
- A Scuba diving regulator to assist the flow of air once it starts flowing.
- Modern vaporizers to optimize efficiency.
- In recoilless rifles Venturi effect is used to decrease the recoil of firing.
- Ventilators.
- Automated pool cleaners.

Q. Can you give a brief description of the Venturi mask?

Ans. It is a high-flow, fixed performance device. An air-entrainment mask contains a jet orifice and air entrainment ports and is designed to fit over the patient's nose and mouth. The oxygen is supplied to the mask through narrow-bore oxygen tubing under pressure. When oxygen under pressure is forced through a small jet orifice entering the mask, a negative pressure is created, which causes room air to be entrained into the mask through the apertures in the Venturi barrel. The resultant air-oxygen mixture containing the prescribed O_2 concentration flows into the face piece for patient breathing. Due to the high-flow rates, the excess gas flushes out the expired CO_2 through the holes on the sides of the mask. As a result of this there is no rebreathing or no increase in dead space. When oxygen flow is increased, more air is entrained, to maintain a constant oxygen concentration. The system is called high

airflow with oxygen enrichment (HAFOE). Variations in the size of the entrainment ports determine the FiO_2 and variation in the oxygen flow rate determines the total flow delivered. The entrainment mechanism is based on the principles described by Bernoulli. The FiO_2 can be precisely controlled from 0.24–0.6 (24–60%) at high-flow rates by simply exchanging the interchangeable jet nozzles and adjusting the oxygen flow rate (**Table 3**).

In the Ventimask, the Venturi is part of the mask so that air is entrained into the mask, close to the patient. The masks are color-coded and labeled with the inspired oxygen concentration that they will deliver and the flow of oxygen that is required to achieve this. A known FiO_2 can also be delivered to patients breathing spontaneously with an endotracheal tube by attaching the Venturi device to the T-piece. Since, the device is away from the patient, they receive the gases at a lower pressure. The patient will continue to receive the set FiO_2 as long as the FGFs exceed the peak inspiratory flow rates.

TABLE 3: Flow rate and FiO_2 with Venturi mask.

FiO_2 provided by the Venturi valve	Oxygen flow to the valve (L/min)	Amount of air entrained (L/min)	Total flow to patient (L/min)
0.24	2	51	53
0.28	4	41	45
0.31	6	41	47
0.35	8	37	45
0.40	10	32	42
0.60	15	15	30

Q. How does one get a fixed FiO_2 with a Venturi mask?

Ans. The Venturi oxygen delivery device has two variables—oxygen flow and the entrainment ratio. The entrainment ratio is set by the manufacturer. Any back pressure or suction will change the entrainment ratio and more air will be entrained. If all of the oxygen that is delivered to the mask takes part in the total flow, it is possible to derive a mathematical equation which will calculate the amount of air entrained. The total gas flow will be the sum of the oxygen flow and the amount of air entrained.

Entrained air flow is given by the equation:

$$\text{Entrained air flow} = \frac{\text{O}_2 \text{ flow from wall} \times (1 - \text{FiO}_2)}{\text{FiO}_2 - 0.2}$$

For example, if the oxygen flow is 10 L/min and FiO_2 0.6, the ratio will be 1:1; i.e., for each liter of oxygen, 1 L of air will be entrained. **Tables 3 and 4** show the effects of different FiO_2 and different flow rates of oxygen on the total gas flows delivered to the patient.

TABLE 4: Total gas flows with variable FiO_2 .

FiO_2 (L/min)	Oxygen flow (driving) (L/min)	Air flow (entrained) (L/min)	Total flow (L/min)
1.0	10	0	10
0.6	10	10	20
0.6	20	20	40
0.5	10	17	27
0.5	15	25	40
0.4	10	30	40
0.24	2	38	40

To get FiO_2 of 1.0 with oxygen flow rates of 10 L/min there should be no air entrainment and the total gas flows delivered to the patient is 10 L/min. If the patient's peak inspiratory flow rates exceed 10 L/min, there will be air dilution and the delivered FiO_2 will be lower. From **Table 4**, we can see that to deliver FiO_2 of 0.6 the oxygen flow rates and the amount of air entrained are the same. If oxygen flow rate is increased to 15 L/min, then to get FiO_2 of 0.6 the air entrainment should be 15 L/min. But if the patient's peak inspiratory flow rates exceed 30 L/min, there will be air dilution which reduces the FiO_2 actually received by the patient. Furthermore, the group of patients who need a high FiO_2 are likely to have peak inspiratory flows higher than normal. Generally, oxygen flow of 10 L/min are sufficient to deliver an FiO_2 of 0.4 as the peak inspiratory flow rate of 40 L/min usually matches with the total flows going to the patient. While designing the Ventimask it must be taken into account that any change in downstream resistance will reduce the air entrainment which in turn will reduce the total flows being delivered to the patient. Also there must be sufficient space available beyond the Venturi to allow mixing of driving and entrained gas. Therefore, a Venturi from one device may not function as expected if fitted to another manufacturer's product.

Q. Can you describe any medium dependency system?

Ans. Medium dependency oxygen delivery devices are used when the patient needs some amount of respiratory assistance along with oxygen supplementation. It can only be used in spontaneously breathing patients. For example, CPAP mask. The CPAP device consists of a face mask with head strap, O_2 -CPAP valve, flow generator, and wide-bore tubings. The masks are available in various sizes and cover the mouth and nose. The mask is secured with a harness. O_2 -CPAP valves provide CPAP from 2.5 to 20 cm of water. The flow generator can be plugged into wall outlet for oxygen. It has an ON switch, as well as a switch for adjusting the oxygen flow and oxygen concentration. Wide-bore tubing connects the flow generator to the mask. There are two

one-way valves present in the body of the face mask. One is the inlet valve and other is the outlet valve to which the CPAP valve is attached. The facemask, CPAP valve, and the wide-bore tubings are for single use only.

Q. How do you give oxygen therapy to neonates and infants?

Ans. Enclosure systems are used to provide oxygen therapy to neonates and small infants (**Figs. 5A to F**).

Oxygen hoods: These are transparent enclosures designed to surround the head of the neonate or small infant. It has an opening for the neck and comes in different sizes. Hoods are well-tolerated by infants and allow easy access to chest, trunk, and extremities. Due to limitation of the size, these hoods can be used only in children below 1 year of age. A continuous flow of humidified oxygen is supplied to the hood. The hoods deliver 80–90% oxygen at a flow rate of 10–15 L/min.

Oxygen tents: Transparent enclosures in larger sizes (so-called tent houses or huts) are available for patients who are too big for the hoods. The subject lies in the tent and CO_2 is removed by soda lime and water vapor by calcium chloride. The temperature in the tent is regulated by flowing oxygen and air over ice chunks (to prevent accumulation of heat of exothermic reaction). The tents are designed for children. FiO_2 of 0.6–0.7 can be achieved with flow rates of 10–12 L/min. The air changes 20 times/h. High output nebulizers can circulate cooled mist in the tent.

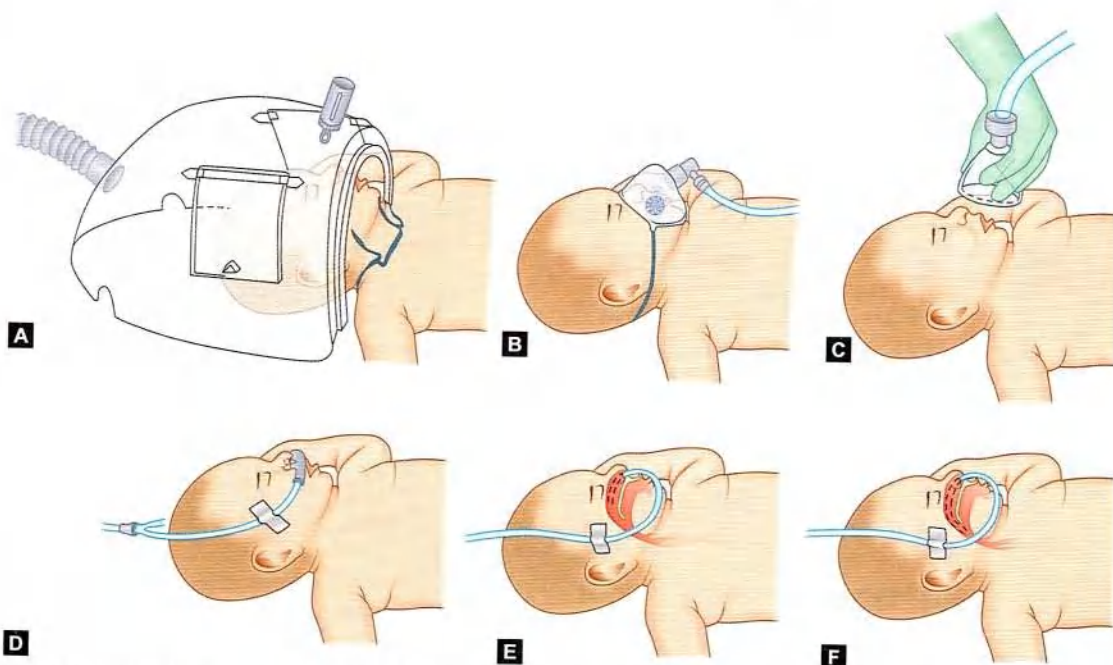
Limitations: As the oxygen concentrations may vary within the hood it should be measured as near the nose and mouth as possible. There is a decrease in oxygen concentration whenever the enclosure is opened. Nasal oxygen may need to be supplied during feeding and nursing care of patients who are confined to hoods. Flows > 7 L/min are required to wash out carbon dioxide:

- Devices can be confining and isolating.
- FiO_2 can vary from 0.21 to 1.0.
- Temperature of the gases in the hood should be maintained to provide a neutral thermal environment.

Closed incubators: They are transparent enclosures that provide a warm environment for small infants with temperature instability. Supplemental oxygen can be added to incubators but may result in an increased oxygen concentration. The primary purpose of an incubator is to provide a temperature-controlled environment. FiO_2 is maintained at 0.4. Humidified oxygen can be provided.

Equipment-related potential harm: Prolonged exposure to humidified oxygen may increase risk for cutaneous fungal infection:

- Hypoxia or hypercapnia can result from inadequate or loss of gas flow.
- Temperature within enclosures should be closely monitored to reduce the potential for cold stress or apnea from overheating in neonates.
- Use of an improperly sized hood can result in irritation of the infant's skin.



Figs. 5A to F: (A) Headbox oxygen; (B) Facemask; (C) Oxygen administration by holding an oxygen source near the infant's face; (D) Nasal cannulae; (E) Nasal catheter; and (F) Nasopharyngeal catheter.

Q. What is nasal high flow (NHF) oxygenation and Optiflow™?

Ans. Apart from traditional oxygen therapy, noninvasive ventilation and invasive mechanical ventilation, recently, a patient with the respiratory failure can be treated with a NHF oxygenation. NHF allows the delivery of up to 60 L/min of heated and humidified gas via a wide-bore nasal cannula. It has got a blender which allows operator to choose the required FiO_2 and thus prevents oxygen toxicity. This unique wide-bore nasal cannula (Optiflow™) is made of soft, flexible, thermoplastic elastomer, the cannula prongs and base sit comfortably under the nose. It delivers gas flow directly into nares without gas jetting. It has adjustable head strap which comfortably fits over the patient's ears. Optiflow™ was demonstrated to generate low level of positive pressure when used at 35 L/min of gas flow and thus may help in avoiding reintubation in select cases. In patients with nonhypercapnic acute hypoxemic respiratory failure, treatment with high-flow oxygen, has shown to reduce 90-day mortality.

Q. How would you clean and sterilize the oxygen therapy devices?

Ans.

Low-flow systems: Under normal circumstances, low-flow oxygen systems do not present clinically important risk of infection and do not require routine replacement on the same patient:

- Nasopharyngeal catheters should be changed every 24 hours.
- Transtracheal catheters should be changed every 3 months.
- *Reservoir systems:* Under normal circumstances, reservoir systems do not present clinically important risk of infection and do not require routine replacement on the same patient.

High-flow systems: Large-volume nebulizers should be changed every 24 hours when applied to patients with an artificial airway.

Enclosure systems: There is no recommendation regarding the frequency of changing oxygen hoods and reservoirs while in use on the same patient.

Q. What factors decide the choice of an oxygen therapy device?

Ans. The need of an oxygen therapy device is determined by measurement of inadequate oxygen tensions and saturations by invasive or noninvasive methods and/or the presence of clinical indicators. Supplemental oxygen flow should be titrated to maintain adequate oxygen saturation as indicated by pulse oximetry (SpO_2) or appropriate arterial or venous blood gas values.

- Nasal cannulae and nasopharyngeal catheters are used to provide low level supplemental oxygen. They are also used to supplement oxygen in patients who are being fed so that there is no interruption in oxygen supplementation.
- Simple oxygen masks are used to provide supplemental oxygen in the moderate range (0.35–0.50, depending on size and minute ventilation) for short periods of time (e.g., during procedures, for transport, in emergency situations).
- Air-entrainment nebulizers can be used when high levels of humidity or aerosol are desired.
- Partial rebreathing masks are used to conserve the oxygen supply when higher concentrations ($\text{FiO}_2 > 0.4$, < 0.6) are warranted (e.g., during transport).
- Nonrebreathing masks are used to deliver concentrations > 0.60 .
- Venturi masks are used when precise FiO_2 has to be delivered, e.g., chronic obstructive pulmonary disease (COPD).

Hoods are used to provide:

- Controlled FiO_2 in infants and small children
- Controlled FiO_2 and/or increased heated humidity to patients who cannot tolerate other devices
- Controlled FiO_2 when the chest, abdomen, and extremities must be accessible to caregivers.

MANUAL RESUSCITATORS**Q. What are the uses of manual resuscitators?**

Ans. These are portable manual ventilating devices used for ventilating the patient during:

- Resuscitation and other critical situations
- Transport of the patient
- As a standby measure for nonfunctioning of anesthesia machine
- Administering anesthesia when anesthesia machine is not available, e.g., in field situation
- For manual ventilation during magnetic resonance imaging.

Q. What are the various types of resuscitators that you know of?

Ans.

- *Laerdal resuscitator bag:* Most commonly used
- The Ambu bag resuscitator, Denmark
- Cardiff infant bag
- Samson-Blease bag
- Sanjivani adult resuscitator
- The air-viva resuscitator.

Q. What does AMBU stand for?

Ans. Artificial Manual Breathing Unit or Air Mask Bag Unit.

Q. Is there a standard resuscitator for all patients?

Ans.

- No. There is no standard resuscitator for all patients. They are supplied in three sizes:
 - *Adult*: It has a capacity of 1,600 mL and is capable of delivering tidal volume of 600 mL. It can be used in adults weighing >30 kg.
 - *Child*: They have the capacity of 500 mL and can be used in patients from weight between 7 and 30 kg.
 - *Neonatal*: They are capable of delivering the tidal volume up to 20–50 mL and can be used in infants up to 7 kg.
- *Maximum tidal volume that can be delivered*: Adult—1,000 mL, child—350 mL, infant—205 mL.
- *Expiratory resistance*—approximately 0.045 cm H₂O/L/min. The expiratory resistance is lower for flows < 10 L/min.
- *Inspiratory resistance*—approximately 0.05 cm H₂O/L/min.

Q. Describe the various components of a manual resuscitator.

Ans.

Design and Function

Manual resuscitators have a compressible self-expanding bag, a bag refill valve, and a patient intake valve (nonrebreathing valve). Optional components include a pressure-limiting device, reservoir bag with demand valve, positive end-expiratory pressure (PEEP) valve, and mechanism for scavenging expired gases (Fig. 6).

Self-inflating Bag

This is oval, cylindrical or sometimes shaped-like a baseball. It is made up of silicone and is cream-white in color. The bag is strengthened either by making its wall thicker, or incorporating circular “ribs” of identical material during manufacture, or by lining it with thick foam so that in the resting state it is expanded. The respirable gas inlet

mechanism is housed at one end and the nonrebreathing valve at the other end. Some bags collapse like an accordion during storage. The bag expands during expiration. If the volume of oxygen from the delivery source is inadequate to fill the bag, the difference is made up by intake of air. The rate at which the bag reinflates will determine the maximum respiratory rate. The size of the operator’s hands determines the actual tidal volume going to the patient and if the hands are small then it can cause hypoventilation. With prolonged exposure to cold conditions, the cycling rate of the bag may be reduced and the unit may become incapable of delivering satisfactory ventilation.

The respirable gas inlet: It has several components:

- *Bag refill (intake) valve*: It is a one-way flap valve which is fitted to the inlet of the self-inflating bag. When the bag is squeezed, the gas pressure inside the bag causes the flap valve to close. This prevents the escape of gas back through the inlet. When the bag is released, its self-inflating characteristics cause entrainment of fresh gas from the respirable gas inlet. This may normally be either air, oxygen or a mixture of both.
- *A small bore nipple*: This is mounted on the inlet, to allow oxygen supply to be attached.
- *A wide-bore gas inlet*: This supplies the bulk of gas (either air or oxygen) entering the bag. The final concentration of oxygen delivered depends on the oxygen flow rates and air entrainment.
- *A reservoir system*: The inlet may be fitted with a reservoir system which stores the oxygen supply. Use of a reservoir significantly increases the oxygen concentration in the inspiratory air. With each bag compression up to 100% oxygen can be delivered to the patient if the oxygen flow rates are greater than the volume given to the patient. The reservoir assembly for the resuscitators consists of a valve unit and either an inflatable bag or an elongated wide-bore hose (popular in pediatric resuscitators). The size of the reservoir may limit the oxygen delivered. If the volume of the reservoir is less than that of the bag, the inflowing oxygen may not be sufficient to make up the difference and room air will be drawn in. A large

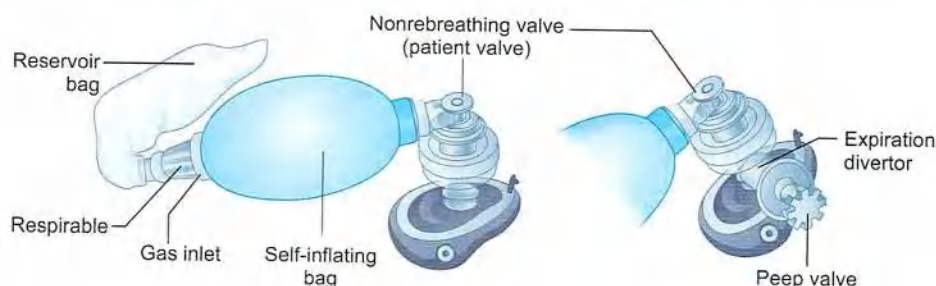


Fig. 6: Manual resuscitator.

reservoir makes the resuscitator more cumbersome. The reservoir assembly is attached to the wide-bore connector in the rear of the intake valve. The valve unit consists of an overflow valve to prevent overfilling due to high flow of oxygen and an entrainment valve to entrain air if the bag collapses due to underfilling.

When the bag is not filling, i.e., during squeezing and in the fully expanded state, the oxygen inflow will initially partly fill the ventilation bag and partly flow out through the open end of the intake valve and fill the reservoir bag. During bag re-expansion there is negative pressure in the ventilation bag which causes the inlet valve to open and oxygen from the reservoir enters the bag.

If the ventilation bag is not squeezed, and the mask is not held tightly over a patient's face, oxygen will flow continuously through the lip membrane of the patient valve. However, if the mask is held tightly in place, or when patient valve is connected to a tracheal tube, excess oxygen will escape through the outlet membrane of the reservoir valve until the ventilation bag is squeezed. During ventilation if the reservoir bag remains flat, it indicates that there is no oxygen supplementation.

Demand Valve

A demand valve connecting a compressed gas source to the self-expanding bag will consistently provide a high inspired oxygen concentration. A negative pressure in the bag triggers a flow of oxygen, which stops at the preset pressure.

A demand valve provides warning of supplemental oxygen. Should the demand valve become stuck or the oxygen supply depleted, the bag will not refill.

Patient Valve (Nonbreathing Valve)

It is also called as direction control valve. The valve should have a transparent housing which will enable the user to check the valve function during use. The various components of the valve ensure that during the inspiratory phase the gas flows from the bag into the patient port only and during expiratory phase the exhaled gases escape from the expiratory port without mixing with the fresh gas stored in the bag.

The inspiratory port is the opening through which gas enters the valve from the bag. Expiratory port is the opening through which exhaled gases pass to the atmosphere. The expiratory port may have a tapered 30 mm connector for attachment of the transfer tube of a scavenging system. The ASTM standard requires that such a connector have ridges in its internal lumen so that it cannot accept a 22 mm male connector. The patient valve has a standard 15 (ID)/22(OD) mm patient end which connects to all standard masks or tube adaptors. The adult masks and the pediatric circular mask size 2 fit outside the patient valve connector. All other (pediatric) sizes fit inside, to reduce the dead space.

During inspiration, as the self-inflating bag is pressed, the valve directs the gas from the bag to the patient. At the same time, expiration port is blocked. When the squeezing of the bag is stopped and the bag is released, the expiration port opens and allows exhalation of the gases to the atmosphere. The valve may have means to prevent air intake so that a spontaneously breathing patient will inhale only from the bag. A patient who breathes spontaneously can inhale oxygen through the resuscitator with minimal resistance. The capacity of the valve is 7 mL.

Inspiration pressure regulator: The infant and child resuscitators have a patient valve with a special pressure limiting device mounted on the upper valve housing. It is also called as pressure relief valve or pop-off valve. This device is usually in the form of spring-loaded disk with the tension on the spring adjusted so that it opens at a desired pressure. It opens at an opening pressure of 45 cm of water for infant and pediatric bags. It prevents lung injury because of barotrauma and also prevents gastric inflation. In adults, the opening pressure is kept at 60 cm of water. One can override this mechanism by either placing a finger over it or fixing it with adhesive tape.

Expiration Diverter

An expiration diverter can be snapped onto the patient valve. The diverter provides an airtight seal to the valve housing but does not prevent the swivel function (possibility of horizontally rotating the bag without interfering with the position of mask or tube) of the valve connector.

A PEEP valve or equipment for measuring, scavenging or monitoring expired gases can be attached to the standard (30 mm OD) outlet port of the diverter.

Q. What is the FiO₂ delivered to a patient using a Laerdal resuscitator?

Ans. When used with oxygen the manual resuscitator is a high-flow high oxygen device. They can be used with or without supplemental oxygen. Concentrations delivered to the patient depend on oxygen flow rate, use of a reservoir, and ventilation technique, e.g., tidal volume, ventilation frequency, time relations during compression—release cycles. The higher minute volume and greater the I:E ratio, the lower the delivered oxygen concentration. The amount of oxygen concentration can be increased by increasing the oxygen flow rate. However, the maximum oxygen that can be delivered by a flow meter is 15 L/min. Oxygen concentration can also be increased by attaching reservoir tubing to the fresh gas inlet where the excess oxygen is stored.

Wastage of oxygen can be avoided by adjusting the oxygen flows so that the reservoir bag does not fill completely during ventilation cycles. If 100% oxygen is desirable, best oxygen economy is obtained when flow is

regulated so that reservoir neither fills fully nor empties completely during ventilation cycles.

Tables 5 to 8 show typical oxygen concentrations that are delivered under a variety of conditions with two popular makes of resuscitators. The differences reflect the size of the relevant reservoirs used.

For delivered oxygen concentrations in Laerdal resuscitator see below:

- **Adult:** Ventilation bag volume—1,600 mL and reservoir bag volume—2,600 mL.
- **Child:** Ventilation bag volume—500 mL and reservoir bag volume—2,600 mL.
- **Infant:** Ventilation bag volume—500 mL and reservoir bag volume—2,600 mL.

Oxygen concentrations (%) in the Ambu system with reservoir.

TABLE 5: Delivered FiO₂ with Laerdal bag (adult).

O ₂ flow (L/min)	Tidal volume (mL) × bag cycling rate per minute					
	O ₂ concentration (%) using reservoir (without reservoir in brackets)					
	500 × 12	500 × 24	750 × 12	750 × 24	1,000 × 12	1,000 × 24
3	56 (37)	39 (32)	47 (33)	34 (29)	41 (32)	30 (28)
5	81 (52)	52 (38)	62 (41)	42 (33)	52 (39)	38 (31)
10	100 (73)	84 (48)	100 (56)	65 (42)	84 (55)	53 (39)
12	100 (84)	97 (53)	100 (61)	74 (45)	94 (60)	59 (42)
15	100 (89)	100 (59)	100 (69)	86 (48)	100 (69)	66 (44)

TABLE 6: Delivered FiO₂ with Laerdal bag (pediatric).

O ₂ flow (L/min)	Tidal volume (mL) × bag cycling rate per minute			
	250 × 20		100 × 30	
	Oxygen concentrations (%)			
	With reservoir	Without reservoir	With reservoir	Without reservoir
10	100	75	100	90

TABLE 7: Delivered FiO₂ with Laerdal bag (infants).

O ₂ flow (L/min)	Tidal volume (mL) × bag cycling rate per minute			
	40 × 30		20 × 40	
	Oxygen concentrations (%)			
	With reservoir	Without reservoir	With reservoir	Without reservoir
4	98	89	98	98

TABLE 8: Delivered FiO₂ with Ambu bag.

O ₂ flow (L/min)	Ventilation volume (mL) × frequency			
	250 × 12	600 × 12	750 × 12	1,000 × 12
2	74	43	38	34
5	100	76	65	54
10	100	100	100	87
15	100	100	100	100

Q. Can you give PEEP with a Laerdal resuscitator?

Ans. Yes. A PEEP valve can be attached to the outlet port of the expiration diverter. The expiration diverter can be attached to the patient valve.

Q. What is the functional analysis of the manual resuscitator?

Ans. The Laerdal valve is the commonly used valve. It contains two unidirectional valves, a fishmouth valve which ensures unidirectional flow from the bag to the patient, and a disk membrane which ensures flow from patient to atmosphere.

Inspiration: Bag compression causes positive pressure in the ventilation bag. This opens the lip-shaped membrane in the patient valve, and also closes the intake valve.

Spontaneous inhalation: This causes negative pressure in the patient valve. This closes the external disk membrane, to prevent inappropriate intake of ambient air.

Exhalation: Whether spontaneous or passive, exhalation causes positive pressure in the front part of the patient valve. This closes the lip membrane, preventing expired air from entering the bag. At the same time, the external disk membrane is lifted away from its seat, allowing expired air to escape to atmosphere.

Bag refill (intake) valve is a one-way flap valve which is fitted to the inlet of the self-inflating bag.

Open: Bag re-expansion or inhalation efforts of a spontaneously breathing patient cause's negative pressure in the ventilation bag. This lifts the disk membrane from its seat, leaving the intake ports open so that ambient or oxygen-enriched air will be drawn into the bag.

Closed: The intake valve stays closed with neutral or positive pressure in the ventilation bag. This occurs when the bag has re-expanded fully, is being compressed, or when no inhalation effort is made by the patient.

Q. How do you sterilize a manual resuscitator?

Ans.

Decontamination

Washing and rinsing is the first step in the decontamination process. Disassemble all the parts and rinse in lukewarm water. Then wash the parts in warm water using a detergent that is compatible with the resuscitator material. Rinse all the parts thoroughly in clean water until all traces of detergent is removed. Allow to dry.

Disinfection/Sterilization

The sterilization methods apply to all parts except reservoir bags, head straps, storage pouch and containers. The high level disinfection methods apply to all parts (**Table 9**).

TABLE 9: Disinfection/sterilization.

	Method	Process parameters		Post-treatment
		Parameters/concentrations	Exposure time	
Sterilization	Steam autoclaving	Autoclave at 132–135°C	15 minutes	Allow parts to cool
	Ethylene oxide	Temperature 55°C, 30–80% relative humidity, ethylene oxide concentration 630 mg/L	180 minutes	Aeration time: 24 hours ambient temperature
High level disinfection	Glutaraldehyde (Cidex)	Concentration 2% Ambient temperature	60 minutes	Rinse all parts in sterile water and dry
	0.5% sodium hypochlorite	Concentration 0.5% Ambient temperature	20 minutes	Rinse all parts in sterile water and dry

Q. How would you check the valve functions to ensure proper operation of the resuscitator after each disassembly-reassembly?

Ans. Test valve functions to ensure proper operation of the resuscitator after each disassembly-reassembly. A test lung is required to complete the test procedures.

Intake Valve (Bag Refill Valve)

Compress the ventilation bag with one hand and close its neck opening with your other hand. Release the grip on the bag. Rapid bag re-expansion confirms efficient air intake.

Close the neck opening and try to compress the bag. If the bag cannot be compressed with reasonable force, or if bag compression forces the air out between your hand and neck of the bag, the valve efficiently prevents backward leakage of air.

Patient Valve (Nonrebreathing Valve)

Ensure that the patient valve is properly assembled. Attach the patient valve to the bag. Hold a reservoir bag over the patient port. Ensure a tight seal between the patient port and reservoir bag and that the external disk membrane is not blocked. Compress the bag with your other hand several times. Inspect that the lip valve opens during compression. Filling of the reservoir bag in this set-up confirms that the patient valve efficiently directs air to the patient.

With the filled reservoir bag held firmly to the valve connector, compress the reservoir bag while watching the external disk membrane mounted on the outside of the patient valve housing. Lifting of the disk membrane from its seat confirms that air is correctly directed to atmosphere instead of being returned to the ventilation bag.

Patient valve with pressure relief valve: Close patient port connector with your thumb while compressing the bag several times. Visual and audible opening of the relief valve confirms its operation.

Oxygen nipple in the respirable gas inlet: After compressing the ventilation bag, close the reservoir valve connector on the intake valve and then release the bag compression.

There will be slow re-expansion of the bag which confirms that the oxygen nipple is open.

Reservoir flap valves (located in the reservoir valve assembly).

Attach a reservoir bag to the reservoir valve assembly. Close port of the reservoir valve and press on the reservoir bag. Compression of the reservoir bag and visual rise of the outlet flap valve confirms that the reservoir valve efficiently vents excessive gas to atmosphere.

With the patient valve in place and the reservoir attached to the reservoir valve assembly, perform several compression-release cycles on the ventilation bag until the reservoir bag is flat and empty. Rapid re-expansion of the ventilation bag after flattening of the reservoir bag confirms that the reservoir valve efficiently lets in ambient air to compensate for lack of gas in the reservoir or insufficient gas flow through the oxygen tubing and nipple.

Q. What are the possible hazards during the use of a manual resuscitator?

Ans.

- *High airway pressure:* If excessive force is used to compress the bag in intubated patients.
- Barotrauma due to use of high tidal volume.
- Hypocapnia due to hyperventilation.
- Rebreathing can occur if the nonrebreathing valve is stuck in the inspiratory position or is improperly assembled.
- Failure of the pressure limiting device in pediatric resuscitator.
- Hypoventilation if sufficient force is not applied to the manual pressing.
- Low delivered oxygen concentration may result due to insufficient oxygen flow, detached or defective oxygen tubing or problems with the oxygen enrichment device.
- Risk of infection due to improper sterilization.

Q. What are the differences between the Laerdal, Ambu, and Air Viva resuscitator?

Ans. The differences between Laerdal, Ambu, and Air Viva resuscitator are summarized in **Table 10**.

TABLE 10: Comparison of Laerdal, Ambu, and Air Viva resuscitator.

<i>Laerdal resuscitator</i>	<i>Ambu</i>	<i>Air Viva</i>
It is made up of silicone, is cream-white in color, and is oval in shape. The bag is strengthened either by making its wall thicker, or incorporating circular "ribs"	It is a black-colored self-inflating double-ended bag, with foam rubber lining	It is green colored, self-inflating single-ended bag
Available in three sizes—adult, child, and neonate	Available in two sizes—adult and baby	
Laerdal nonbreathing valve is attached to the front of the bag	Ambu E2 nonbreathing valve is attached to the front of the bag	It has an expiratory valve with a common port for inspired and expired gases. Bag refilling occurs during the expiratory phase as a result of which the expired gases get drawn in resulting in partial rebreathing. Oxygen nipple in the front
Distal end has air inlet valve and oxygen inlet connection with or without attached oxygen reservoir	Distal end has air inlet valve and oxygen inlet connection with or without attached oxygen reservoir	It is closed distally
It can be used for anesthesia purpose		It cannot be used for anesthesia purpose. Not advisable if prolonged resuscitation is required

SUGGESTED READING

1. Davey AJ, Diba A. Ward's Anaesthetic Equipment, 5th edition. Philadelphia, PA: Elsevier Saunders; 2005.
2. Dorsch JA, Dorsch SE. Understanding Anesthesia Equipment, 5th edition. Philadelphia, PA: Lippincott Williams and Wilkins; 2008.
3. Ely J, Clapham M. Delivering oxygen to patients. BJA CEPD Reviews. 2003;3:43-5.
4. Parke R, McGuinness S, Eccleston M. Nasal high-flow therapy delivers low level positive airway pressure. Br J Anaesth. 2009;103(6):886-90.

Videolaryngoscopes

Atul Prabhakar Kulkarni, Madhavi Desai, Shilpushp Jagannath Bhosale

■ INTRODUCTION

Fiberoptic intubation is the gold standard in the management of an anticipated difficult airway. There is no standard definition of difficult intubation. According to the American Society of Anesthesiologists (ASA), difficult intubation is defined as need for more than three attempts to intubate in 10 minutes. As per the Canadian Society of Difficult Airway, intubation was considered difficult when it is not possible to visualize any portion of vocal cords with conventional laryngoscopy or when the intubation requires more than one attempt, a change in blade, an adjunct to direct laryngoscopy (DL), or use of an alternative device. Cormack-Lehane grades III and IV with conventional DL have been demonstrated to be associated with difficult intubation. In the literature, 5–6% of the intubations in operating room and 16% intubations outside operating room are reported as unanticipated difficult intubations.

Videolaryngoscopy (VL) is undoubtedly one of the major advances in practical anesthesia in last two decades. In 2001, Canadian surgeon, John A Pacey was the first to embed a miniature video chip (complementary metal oxide semiconductor) into a modified Macintosh laryngoscope—the GlideScope videolaryngoscope.

Q. What is a videolaryngoscope?

Ans. Videolaryngoscopes are indirect laryngoscopes that work on the principle of “looking around the corner” which is made possible by placing the camera at the distal tip of the laryngoscope blade.

Videolaryngoscope consists of a handle, laryngoscope blade, and a micro video chip camera embedded into the end of the blade. The image of the laryngoscopic view is transmitted to an external monitor, liquid crystal display (LCD) through a video system. Thus, videolaryngoscopes provide a wider viewing angle, making alignment of the oral, pharyngeal, and tracheal axes less mandatory. It allows the operator to perform tracheal intubation while watching the video screen instead of looking through the small opening of the mouth.

The indirect optical laryngoscopes are often called videolaryngoscopes because these laryngoscopes generally use video technology, so the image of the glottis is transmitted to a remote video screen.

Q. What is the working principle of the videolaryngoscopes?

Ans. *Direct laryngoscopy (DL)* is the direct visualization of vocal folds and the glottis. The conventional direct laryngoscope uses a line of sight provided by a rigid viewing instrument with a light on the blade or intraoral portion, which requires a direct view of the larynx. DL requires anatomical alignment of the oral, pharyngeal, and laryngeal axes with the line of sight view (**Fig. 1**).

Indirect laryngoscopes allow the operator to visualize the virtual image of patient's vocal cords by a means other than obtaining a direct line of sight. For the purpose of intubation, this is facilitated by fiberoptic bronchoscopes, videolaryngoscopes, fiberoptic stylets, and mirror or prism optically enhanced laryngoscopes.

Because of “Looking around the Corner” method, the greater the curvature of the blade, the greater the refracted angle and the more curved would be the trajectory of the endotracheal tube (ETT).

Thus, devices with blades with hyperangulated curves might provide better visualization of the anterior larynx, but might make the placement of the ETT more difficult compared to devices with lesser blade curvature.

Q. How are the videolaryngoscopes classified?

Ans. Videolaryngoscopes are classified in various categories depending on several features:

- *Method of tube advancement:*
 - Videolaryngoscopes with integrated tube channel, e.g., Airtraq, Pentax airway scope, and King Vision
 - Videolaryngoscopes without integrated tube channel, e.g., GlideScope, McGRATH, and C-Mac.

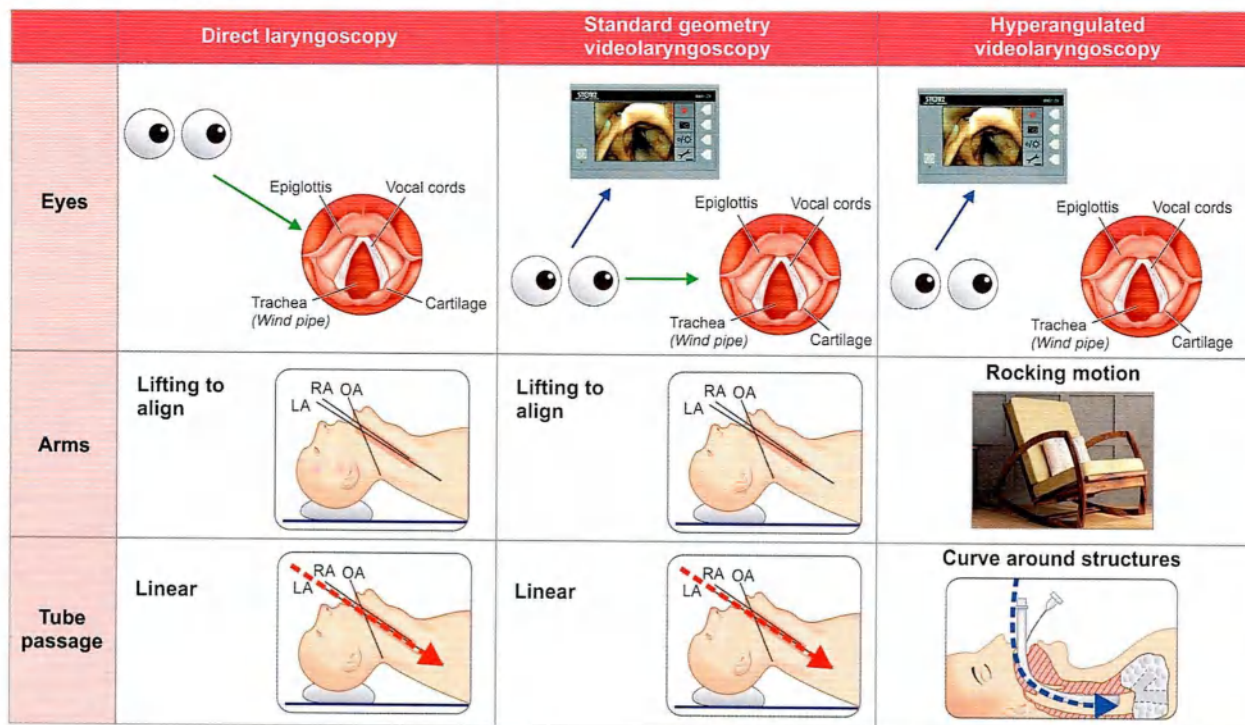


Fig. 1: Direct laryngoscopy—line of sight.

- *Depending upon the curvature of the blade:*
 - Videolaryngoscopes with standard Macintosh blades, e.g., V-Mac and C-Mac.
 - Videolaryngoscopes with angulated blades, e.g., McGrath series 5 videolaryngoscope, GlideScope videolaryngoscopes (GlideScope original, Ranger, and Cobalt), and C-Mac with D blade.
 - *Optical laryngoscopes:* Optical laryngoscopes can be equipped with a video camera and thus function as videolaryngoscopes with a remote screen, e.g., Truview, Airtraq optical laryngoscope, and Bullard laryngoscope.
- Q. What are the advantages of videolaryngoscope?**
- Ans.*
- Display systems of the videolaryngoscope give large, bright, and higher resolution image as compared with the small monocular image obtained from fiberscopes.
 - Because of the design of many videolaryngoscopes, the anterior angulation of the camera enables the operator to “look around the corner” which is not possible with conventional DL.
 - In almost all the videolaryngoscopes, the anatomy of the blade is such that there is a steep bend for alignment of oral, pharyngeal, and laryngeal axes for viewing the glottis. Also, the POGO (percentage of glottic opening) score is better than the Cormack-Lehane classification of laryngeal appearance.
 - The videolaryngoscope improves the Cormack-Lehane grade of visualization of glottis and navigation or advancement of ETT. Thus, less or no pressure is exerted on the upper airway structure with videoscopes.
 - Videolaryngoscopy allows other healthcare providers to visualize the anatomy that the operator is viewing. Thus, the instructor can guide the trainee operator during procedure, making these devices extremely useful in teaching and they may replace “over the shoulder” laryngoscopy teaching in the future.
 - The assistant can help facilitate intubation by providing external laryngeal manipulation to provide optimal visualization of cords as he is himself able to see the field of vision on the monitor. The assistant can determine the amount of cricoid pressure for the facilitation of tube insertion.
 - Stress during the intubation process is less since assistant and performer both can see the intubation.
 - It is possible to record the procedure for records and teaching:
 - Esophageal intubation cannot only be avoided but also picked up immediately by the instructor having a good screen view of the larynx when the trainee is performing tracheal intubation using a videolaryngoscope.
 - Instead of a sniffing position or neck extension, a more neutral head position is recommended, and a

midline blade insertion is advised leading to less neck manipulation.

Movement of the cervical spine is less during videolaryngoscopy as compared to Macintosh laryngoscope, as there is no need to align the oral, pharyngeal, and tracheal axes. For the same reason, significantly less force is exerted on the maxillary incisors during videolaryngoscopy, minimizing potential for dental damage.

Appropriately chosen videolaryngoscopes are beneficial for the management of both anticipated and unanticipated difficult laryngoscopy. The force required when intubating with a videolaryngoscope is less than that required for DL, resulting in less risk of trauma to soft tissues and teeth, and a reduced incidence of sore throat.

Q. Should videolaryngoscopes replace conventional laryngoscopes as first-line devices?

Ans. Although first-line use of videolaryngoscope has not yet been recommended, several videolaryngoscopes have a higher rate of successful intubation when used as a rescue device when DL fails. As most difficult intubations are not anticipated, first-line use of videolaryngoscopy not only reduces the risk of difficulty, but, when this occurs, eliminates the need for the intubator to swap to another device when time and oxygenation are critical. The number of attempts at laryngoscopy can be kept to a minimum, and it is highly likely that unanticipated difficult intubation would be less frequent if videolaryngoscopes were used as a first-line technique.

Q. What are the limitations of videolaryngoscopes?

Ans. Videolaryngoscopes require additional training to develop the psychomotor skills for hand-eye coordination necessary for the correct handling of the ETT while looking at the screen.

Videolaryngoscopy (VL) often allows a good view of the glottis, even in patients who have Cormack-Lehane III and IV grade views with conventional laryngoscopes. However, despite good view of the laryngeal inlet with videolaryngoscope, endotracheal intubation may prove to be difficult. This is because though view is obtained "around the corner," intubation still needs some alignment of axes, as the ETT keeps going posteriorly unless preshaped stylets are used.

The insertion of the styleted ETT into the mouth should be visually followed until it appears on the screen, and never advanced blindly to limit oropharyngeal trauma.

Certain principles that need to be understood and followed regarding the use of videolaryngoscopes are:

- Experience with a standard Macintosh laryngoscope does not equate to skill with a videolaryngoscope or experience with one type of videolaryngoscope does not equate to skill with all videolaryngoscopes.
- A good videolaryngoscopic view of the vocal cords does not guarantee easy intubation, in fact many a times, a

suboptimal view of the glottis facilitates easy intubation. Hence, it is necessary not to go too close to the glottis or too deep into the vallecula.

- Bougie may not be the best option when there is difficulty, and in fact a styleted tube following the curvature of the blade is required.
- The choice of videolaryngoscope must be according to indication and anticipated difficulty and one size fits all may not always be the best option.

Q. What is the advantage of videolaryngoscopes over fiberoptic bronchoscope?

Ans. In fiberoptic intubation, when the tip of the bronchoscope is in the trachea, railroading of the ETT over the fiberscope remains a "blind technique" and may result in injury to laryngeal structures where tube impingement commonly occurs. With videolaryngoscopes, the larynx remains in view throughout the intubation process.

Q. What are the limitations of videolaryngoscopes when compared with fiberoptic bronchoscope?

Ans.

- Videolaryngoscopes cannot be introduced through nose or tracheostomy.
- Some space is required for introducing videolaryngoscopes, so these devices cannot be used in patients with complete trismus or wired jaw.
- Videolaryngoscopes are not helpful in confirming the placement of a double lumen tube (DLT), bronchial blocker, or performing pulmonary toilet.

Q. What are the complications of videolaryngoscopy?

Ans. The efficacy of videolaryngoscope devices is usually judged by outcome measures, such as intubation success rate, time to intubation, and best laryngeal view obtained.

Various studies have shown that "time to intubation" is significantly longer with videolaryngoscopes. The studies conducted even in children with normal airways, videolaryngoscopic intubation takes longer than intubation with DL. This increases the risk of hypoxemia in children and also creates stress to secure the airway. Other issues are prolonged apnea time, rather than laryngeal manipulation with the laryngoscope, increases the blood pressure and the heart rate and may be more stressful to the patient.

In addition, prolonged apnea with a videolaryngoscope may cause hypoxia in patients with reduced oxygen store, such as obese patients and obstetric women. These patients have minimal tolerance to apnea as they desaturate faster due to reduced functional residual capacity.

The difficult part is to direct the tube toward the glottis and between the vocal cords, which are not in the line of sight. Difficulty during insertion of the tracheal tube may result in injuries to the soft palate, oropharynx, and tonsils, even penetration of the palatal arch has been reported.

In inexperienced hands, in particular, there is possibility of ETT impingement at the vocal cords or luminal surface of the anterior tracheal wall. Persistent threading of the tube results in the tube bending further instead of passing down the trachea. This can often be overcome by rotating the tube at 180° after withdrawing the stylet.

Q. What is the role of videolaryngoscopes in emergency settings and intubations outside operating room?

Ans. The role of videolaryngoscope is expanding beyond anesthesiology, such as in intensive care unit (ICU), emergency room (ER), field resuscitation, and aviation medicine, the gold standard still remains the art of mastering the DL with conventional laryngoscopes.

As per a recent systematic review and meta-analysis published in 2018, there was greater first-pass intubation with videolaryngoscopy compared with DL amongst novice/trainee clinicians, but not amongst experienced clinicians or paramedics/nurses. There was no difference in first-pass intubation with videolaryngoscopy compared with DL during cardiopulmonary resuscitation or trauma. Videolaryngoscopy compared with DL was associated with fewer esophageal intubations.

Based on available evidence from above studies on emergency intubation, it may be concluded that C-Mac videolaryngoscope can offer improved laryngeal views compared with DL, but there are conflicting results about whether the improved laryngeal views may result in increased intubation success rate and decreased intubation time. Visualization of the airway on a monitor may be helpful for less experienced intubators in airway management and possibly decrease the rate of esophageal tube misplacements.

Q. Do videolaryngoscopes have any role in pediatric difficult airway?

Ans. An unanticipated difficult airway is extremely rare in children. Most of the times, it is associated with congenital syndromes such as Pierre Robin, Treacher Collins, Arnold-Chiari, and microcephaly. The history and appearance of the child almost always alert the anesthetist to anticipate a difficult airway.

The meta-analysis of use of videolaryngoscopes in children by Sun et al. demonstrated that although videolaryngoscopes improved glottis visualization in pediatric patients, this was at the expense of prolonged time to intubate. Also, there is no randomized controlled trial using videolaryngoscopes in children having anticipated difficult airway.

Most of the videolaryngoscopes are also useful in older children, only few are available in sizes that may be used in children younger than 2 years of age:

- Airtraq disposable optical laryngoscope
- GlideScope videolaryngoscope
- C-Mac videolaryngoscope

- Truview PCD™ infant
- McGrath videolaryngoscope.

The Airtraq is available in two pediatric sizes. "Infant" (size 0) accommodates tube size 2.5–3.5 and "Pediatric" (size 1) accommodates tube size 3.5–5.5. Both laryngoscopes require a mouth opening of 12–13 mm. Combining the Airtraq with a stylet is not recommended as it increases the risk of airway injuries in children.

The pediatric GlideScope Cobalt has a reusable video baton (handle) and single-use laryngoscopy blades in two sizes. Owing to the 70° angulation of the blade, it can be used only for indirect laryngoscopy.

The C-Mac videolaryngoscope is available in Miller blade size 0 and size 1, Macintosh size 2, and a pediatric C-Mac D blade. A recent Cochrane review comparing videolaryngoscopy versus DL for tracheal intubation in neonates by trainees performing neonatal intubations, highlighted the usefulness of videolaryngoscopy as a teaching tool. This suggested that videolaryngoscopy increases the first attempt of intubation success compared with DL; however, there was no difference in time to intubation, number of attempts for intubation, oxygen desaturation, and airway trauma. Residents reached competency faster with videolaryngoscopy (second intubation) compared with DL (seventh intubation).

McGrath blade size 1 is successfully used in preterm neonates and infants weighing from 0.8 to 5.4 kg.

The handle of the C-Mac is bulky and abuts the chest of small children and therefore the laryngoscopy becomes difficult in small infants.

The Truview PCD™ infant is a small handy laryngoscope, with an eyepiece and optics, which provides a wide and magnified laryngeal view at a 46° anterior refracted angle. The height of the laryngoscope blade is only 8 mm and therefore can be used even in neonates. The side port allows insufflation of oxygen, which significantly prolongs apnea time before desaturation, occurs in neonates.

Q. What is the current status of videolaryngoscopy in the management of difficult airway?

Ans. Meta-analyses of RCTs comparing video-assisted laryngoscopy with DL in patients with predicted or simulated difficult airways report improved laryngeal views, a higher frequency of successful intubations, and a higher frequency of first attempt intubations with video-assisted laryngoscopy (Category A1-B evidence); no differences in time to intubation, airway trauma, lip/gum trauma, dental trauma, or sore throat were reported (Category A1-E evidence). As per the All India Difficult Airway Association (AIDAA) 2016 guidelines on anticipated difficult airway, either DL or VL is to be considered in plan A for intubation.

Q. Describe the various types of GlideScope.

Ans. In 2001, a Canadian surgeon, John A Pacey, introduced the GlideScope videolaryngoscope.

Three types of GlideScope videolaryngoscopes are available: GlideScope original, GlideScope Cobalt, and GlideScope Ranger.

The original GlideScope is reusable. It has a nondetachable curved blade, a handle, 7" LCD monitor, and a camera (Fig. 2A). The laryngoscope blade is made up of high-resistance medical grade plastic and the distal third of the blade angulates upward, at approximately 60°. The maximum width of the blade at any place does not exceed 18 mm.



Fig. 2A: GlideScope.

The GlideScope is introduced from the middle rather than the angle of the mouth, in the oropharynx till epiglottis is visualized. The use of a preshaped stylet, with curvature matching the shape of the blade, is recommended to facilitate intubation. **Table 1** displays comparative features of the three types of GlideScopes.

Disinfection and Sterilization

Autoclaving, ultrasonic cleaning, or pasteurization is not recommended. The GlideScope GVL is a nonsterile, reusable device. The GlideScope cobalt video baton is a nonsterile, reusable device. When used as intended, it is protected from direct contact with the patient by the sterile, single-use GVL Stat (Fig. 2B). Low-level disinfection (70% isopropyl alcohol with minimum 1 minute exposure or sodium hypochlorite 500 ppm with minimum 1 minute exposure) is recommended for the video baton after every patient use. Clean the exterior of the monitor and the video cables, wipe with 70% isopropyl alcohol or bleach, i.e., sodium hypochlorite (100 ppm) or a mild detergent and water.

Q. Describe McGrath laryngoscope.

Ans. The McGrath series 5 videolaryngoscope is an anatomically shaped, disposable videolaryngoscope. It has a video camera embedded into a "camera stick" completely, in such a way that no part of the handle or the camera comes in contact with the patient's mouth.



Fig. 2B: GlideScope STAT.

The single use transparent plastic blade (available in three adult lengths) is 13 mm wide and covers the steel

TABLE 1: The features of three types of GlideScope.

GlideScope	Blade shape and size	Monitor	Portability	Disposability	Antifog mechanism
GlideScope original	Angulated, sizes 2–5	Separate, 7" LCD monitor	-	Reusable	Yes
GlideScope cobalt (GVL Stat)	Angulated, sizes 1–4	Separate, 7" LCD monitor	-	Yes (has a plastic blade which slides over the flexible video baton)	Yes
GlideScope ranger	Angulated reusable: Sizes 3 and 4 single-use sizes: 1–4	Separate, 3.5" LCD monitor	Yes, used in military and prehospital settings	Reusable or disposable	Yes

(LCD: liquid crystal display).

bodied camera stick. The length of the camera stick can be easily increased to a maximum of 34 mm, using a simple ratchet mechanism for different-sized patients (**Fig. 3**). The blades are available in sizes 2, 3, and 4. The resulting video image is displayed on a small-colored LCD (1.7") mounted on top of the laryngoscope. The handle with the camera stick can be disassembled for sterilization. The device is powered with a single 1.5 vAA battery (life of 60 minutes of noncontinuous use) and features a single electronic control: an on/off switch which is located at the top of the unit.

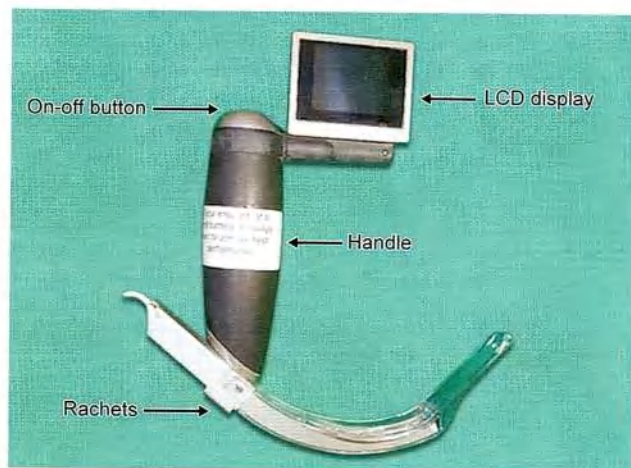


Fig. 3: McGrath videolaryngoscope (LCD: liquid crystal display).

Method of insertion: Introduce the laryngoscope in the midline or slightly to the left (not angle of the mouth). As with other videolaryngoscopes like the GlideScope, a stylet must be used with the ETT, to allow it to be manipulated through the vocal cords.

Advantage: The device is light weight (350 g); therefore, it is easily portable.

Disadvantage: McGrath videolaryngoscope has no video output jack, so the image cannot be seen on a remote screen. Also, it does not have integrated antifogging mechanism.

Q. Describe Storz C-Mac videolaryngoscope in brief.

Ans. In 2003, Kaplan and Berci introduced the Storz C-Mac videolaryngoscope. The C-Mac laryngoscope has a standard Macintosh reusable blade (sizes 2–4) (**Fig. 4A**) with an integrated video camera. In addition, angulated blade is available only in size 4 (**Fig. 4B**) which is called C-Mac “D” Blade. The camera is housed within the laryngoscope handle and the magnified image is displayed on a screen.

In the C-Mac laryngoscope, a fiberoptic bundle is coupled to the internal video camera, directed at approximately 25° from the line of sight. The newer C-Mac direct coupler interface (DCI) laryngoscope is a part of the DCI video intubation system, in which the blades are

interchangeable with flexible fiberoptic and straight Miller blades. C-Mac consists of only two parts, a laryngoscope and a monitor, connected via a single cable. Therefore, it is portable, more robust, and less expensive as compared with the older V-Mac.



Fig. 4A: C-Mac videolaryngoscope Macintosh blade.

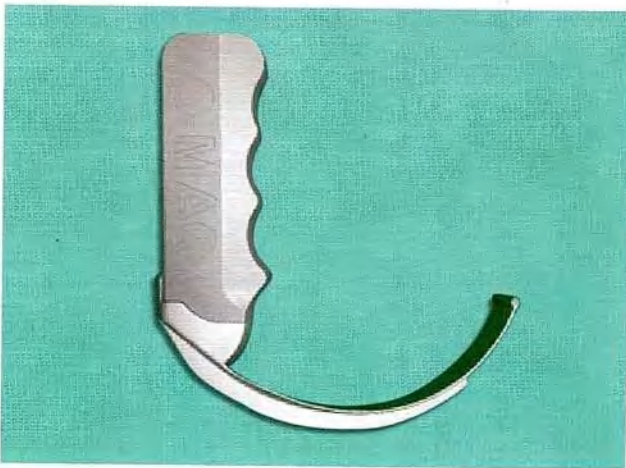


Fig. 4B: C-Mac videolaryngoscope D blade.

Disinfection and Sterilization

Encrustations are removed using an active cleaner and a disinfected using a soft brush. The C-Mac blades and electronic modules are validated for low-temperature preparation methods of sterilization up to a maximum temperature of 60°C. It can be cleaned manually or by using a machine and sterilized with STERRAD (low temperature, hydrogen peroxide gas plasma technology to sterilize instruments and medical devices). At the end of the process, the optical surfaces of the image chip and light-emitting diode (LED) illuminator are rewashed with a cotton tip applicator soaked in 70% isopropyl alcohol. Ultrasound bath and steam sterilization are not recommended.

Q. Describe Bullard rigid fiberoptic laryngoscope.

Ans. The Bullard laryngoscope is an S-shaped rigid fiberoptic laryngoscope with an anatomically curved blade. A sheath is provided for introduction of fiberoptic bundles for illumination and visualization on the posterior aspect of the handle. The handle may contain batteries and a halogen bulb and/or have an adaptor for a flexible cable that is connected to a high-intensity halogen or xenon light source. Light from the source is transmitted to the distal blade through the fiberoptic bundle. It is, therefore, a rigid and fiberoptic laryngoscope together in the same apparatus. A viewing arm with an eyepiece is located at an angle of 45° from the handle (**Fig. 5**).

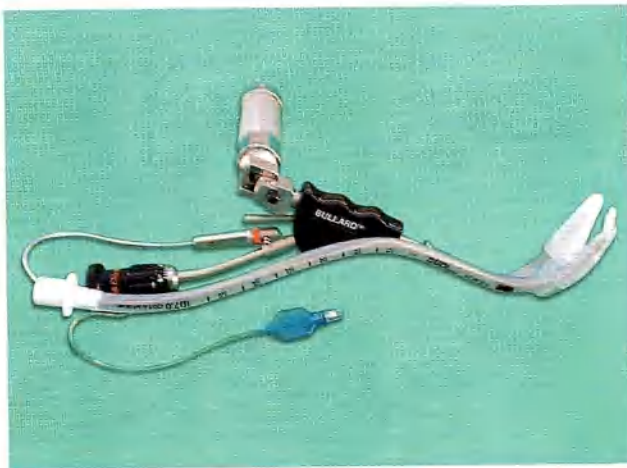


Fig. 5: Bullard laryngoscope.

A working channel for suction and/or insufflation of oxygen extends from the body of the scope to the tip where the light bundles end. The proximal end of the handle has two openings. The first opening is of Luer-taper type of connection. This can be used to connect a Luer-lock connection for equipment such as syringes for medication, local anesthetic, or saline. It can also be used for suction or insufflation of oxygen during the procedure. The second opening has an attachment for the introducing stylet. The introducing or intubating stylet or multifunctional stylet attaches near the base of the viewing arm with a spring-loaded, locking mechanism. It is thin, with a curve to the left at 20° near the tip. This brings the end of the stylet into the field of vision and facilitates passage of the ETT into the laryngeal inlet.

The multifunctional stylet is a long, hollow tube fixed to and parallel to the laryngoscope body. Flexible fiberoptic, tracheal tube exchanger, or a small catheter can be introduced through it. It is more difficult to maneuver than the introducing stylet. The adult tracheal tube is threaded over the lubricated stylet so that the tip of the stylet protrudes through the Murphy eye.

The Bullard laryngoscope is available in three sizes: pediatric, pediatric long, and adult. The adult has a deeper curve than pediatric blades. Also, a plastic tip extender is available to lengthen the tip of the adult blade. This is useful for intubating male patients but is usually not required for female patients. For infants and children and patients with height <5' a pediatric long-sized laryngoscope blade is used. In children >8–10 years of age and patients with height >5' but <6', the adult-sized blade is used. In patients with height >6', an adult blade with tip extender is used (usually, required in male patients).

Advantages: It can be used in patients with a mouth opening as small as 6 mm. Jet ventilation or oxygen insufflation can be given through the working channel. The channel can also be used for suction. Administration of oxygen helps to clear secretions, to prevent fogging, and desaturation. It can be converted into a videolaryngoscope.

Method of Insertion

For oral intubation, ETT is mounted on the stylet. The blade is inserted in the midline of the oral cavity, with the handle horizontal. As the blade is advanced, the handle is rotated to the vertical position so that the blade slides over the tongue. The blade is then elevated against the dorsal surface of the tongue with minimal upward movement exerted along the axis of the laryngoscope. This upward movement will result in the blade of the Bullard laryngoscope lifting the epiglottis, providing complete visualization of the glottis opening. The ETT is advanced over the stylet, and under direct vision, it is pushed into the trachea. The stylet is detached and the scope is gently removed after ascertaining that the ETT is in place. In some cases, the tip of the tracheal tube will impact on the right arytenoid, in such situations, the tube may be rotated 180° on the stylet.

Q. Discuss the Airtraq optical laryngoscope.

Ans. Airtraq was developed and patented by Dr Acha. It is a disposable, optical laryngoscope with two parallel conduits, one optical and the other a tube-guiding channel. A low-temperature battery-operated light is present at the tip of the blade which acts as an antifogging mechanism. The battery life is approximately 1 hour. It is available in adult and pediatric sizes, and a size to accommodate DLT (**Fig. 6**).

To use the Airtraq, the ETT is preloaded in a channel next to the optical pathway, and the device is inserted in oropharynx in midline. When the glottis is visualized, the ETT is advanced into the trachea. The Airtraq is then removed, disengaging the ETT with a lateral movement. If the vocal cords are not visible, it usually means that either the laryngoscope is not in the midline or placed too

posteriorly in the vallecula. In case laryngeal structures are not recognized, withdraw the Airtraq slightly.

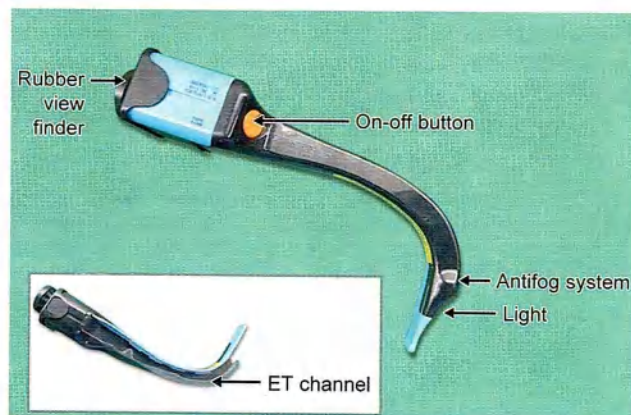


Fig. 6: Airtraq optical laryngoscope.

Advantages: It has an antifog system built into the optical channel which requires 30–45 seconds warm-up time prior to use. It can be converted into videolaryngoscope by attaching optional wireless or wired video system, which should be mounted on top of the Airtraq, after removing the original rubber viewfinder.

Disadvantage: The view through the Airtraq is less panoramic when compared with Macintosh and other videolaryngoscopes. The oral injuries may be unnoticed if they are outside the optical field of Airtraq. **Table 2** describes various sizes and types of blades available with Airtraq.

TABLE 2: Sizes and types of blades of Airtraq.

Description	Endotracheal tube sizes	Mouth opening required for insertion	Use
Regular (size 3—blue)	7.0–8.5	18 mm	Oral intubation
Small (size 2—green)	6.0–7.5	16 mm	Oral intubation
Pediatric (size 1—purple)	3.5–5.5 (cuffed or uncuffed)	12.5 mm	Oral intubation
Infant (size 0—gray)	2.5–3.5	12.5 mm	Oral intubation
Infant nasal (white)	Not applicable	12.5 mm	Nasal intubation
Adult nasal tracheal intubations (orange)	Not applicable	18 mm	Nasal intubation
Double lumen endobronchial tubes	35–41 Fr Standard	19 mm	Double lumen endobronchial tubes

Q. Describe Pentax airway scope in brief.

Ans. The Pentax airway scope has an anatomically shaped polycarbonate blade (PBlade) with a guide channel and a 2.4" LCD monitor built into the top of the handle. The handle houses a built-in charge-coupled device camera and an LED attached to the tip of the scope and a disposable blade powered by two AA batteries lasting approximately an hour. The single use PBlade completely encloses and protects the image tube and camera from oral contamination. The PBlade has two side grooves where the tracheal tube (external diameter between 8.5 and 11.0 mm) and suction catheter (4.0 mm diameter or less) are preloaded (**Fig. 7**).

The ETT is attached to the blade and the tip of the tube is captured on the display screen even before the insertion of the device. Therefore, the location of the tube tip can continuously be monitored during intubation. If the advancement of the ETT is difficult, a tube exchanger or bougie can be passed through the loaded ETT into the trachea and then the tracheal tube from the PBlade can be detached and ETT is railroaded over the bougie.

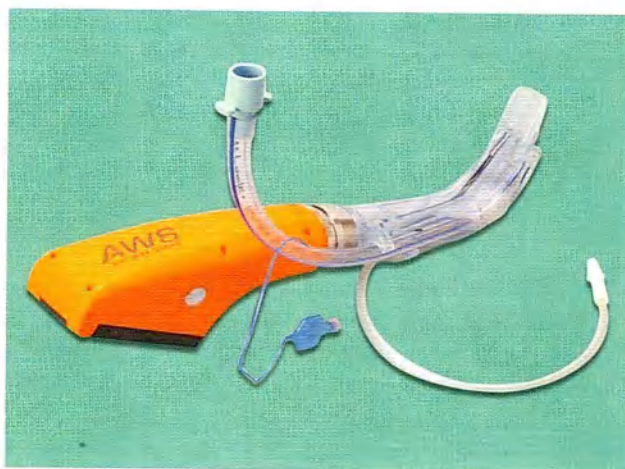


Fig. 7: Pentax airway scope.

Advantages: Pentax airway scope is portable and water resistant. The display screen can be fully rotated to facilitating intubation from a position where the operator is facing the patient. A target mark on the monitor is designed to give direction for advancing the ETT and is intended to facilitate intubation. The PBlade has a separate channel for suction catheter.

Disadvantage: Pentax airway scope is bulky and the length may make its use difficult in patients with large breasts or barrel chest.

Q. Describe in brief the Truview laryngoscope.

Ans. The Truview laryngoscope is a reusable video and optical laryngoscope with a straight blade. It incorporates

a prism and lens assembly. It consists of a combination of optical system with specially profiled 12.8-mm slim steel blade, optical apparatus which provides 42° angled deflection view through 15-mm eye piece (**Fig. 8**).

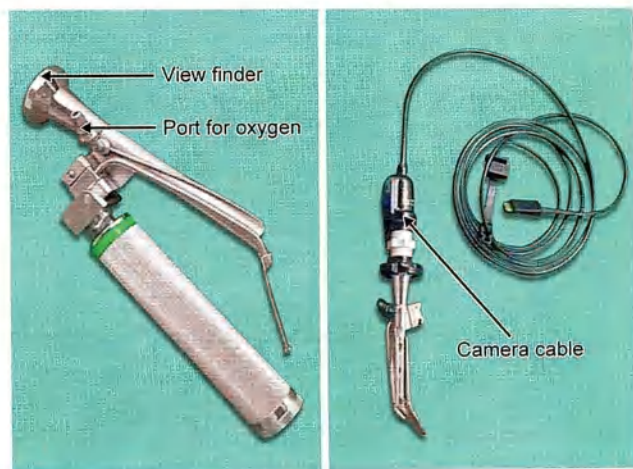


Fig. 8: Trueview laryngoscope.

Method of insertion: The ETT is loaded on the preshaped stylet supplied with the laryngoscope. The laryngoscope is inserted through middle of the mouth and advanced partly into the oropharynx. Then start looking through the viewing lens (or the monitor) and advance while lifting the mandible gently until the glottis is visualized. The trachea is then intubated with the ETT over the stylet and the stylet is withdrawn.

Advantages: The Trueview laryngoscope has an oxygen port on the blade for oxygen insufflation and thus it slows the desaturation of anesthetized patient, also prevents the accumulation of mist and secretions on the lenses, and serves as an antifogging mechanism. Recommended oxygen flows to prevent fogging are up to 5 L/min for blade sizes 0 and 1 and up to 10 L/min for blade sizes 2, 3, and 4. It is light weight and portable device (**Table 3**).

The Trueview laryngoscope blades are available in five sizes (neonate to adult).

It can be converted to a videolaryngoscope by connecting a magnetic camera to the blade and the image is displayed on 5" LCD monitor.

TABLE 3: Patient's age and weight and selection of Trueview laryngoscope blade.

Blade name	Blade mark	Patient's age (years)	Weight range (kg)
Neonatal	0	<1	0.8–4
Infant	1	1–3	4–8
Child	2	3–16	8–60
Adult	3	>16	50–120
Large adult	4	>16	>120

Cleaning, Sterilization, and Disinfection

In Trueview sizes 2, 3, and 4, some components may be disassembled. Trueview blade can be cleaned in its assembled form. The removable components of the blade (the replaceable FiberClip—fiberoptic light guide and the optical view tube) may be disassembled for easier handling during cleaning.

Immediately after use, the Trueview laryngoscope blade should be immersed in water. External surfaces should be gently scrubbed in soapy water with soft brush to remove encrusted deposits. Rinse the blades in demineralized water and dry thoroughly before disinfection and/or storage. Oxygen port can be cleared and/or cleaned using an air or gas jet.

High-level disinfectants such as Cidex Plus (2% glutaraldehyde for minimum 20-minute exposure) and Cidex OPA (0.55% w/w ortho-phthalaldehyde 12-minute exposure) may be used. Chemical detergents containing caustic or alkaline ingredients such as povidone-iodine solutions, peroxide solutions, and sodium hypochlorite are not recommended.

Do not steam autoclave the Trueview blades 0 and 1. If Trueview PCD™ blade sizes 2, 3, or 4 are to be sterilized in a steam autoclave, remove the optical view tube and set aside to be sterilized separately as described above. The remaining parts may be placed in a steam autoclave. Ultrasonic cleaning and "flash" autoclaving are not recommended.

Q. Describe in brief the Bonfils retromolar intubating fibroscope.

Ans. The Bonfils fibroscope was developed 20 years ago for use by ENT (ear, nose and throat) surgeons. The device was introduced in clinical management of patients with difficult airway in mid-1990s. The Bonfils (**Fig. 9A**) is a 40 cm long, semirigid optical stylet with an external diameter of 5.0 mm and a fixed anterior tip curvature of 40°. Its fiberoptic bundle is encased in a stainless steel tube that provides 15,000 pixel resolution and is available with and without a 1.2 mm working channel. The adult stylet can accommodate 6.5 mm or larger ETTs and the pediatric stylet can accommodate 2.5–6.0 mm ETTs. Image of the glottis can be seen through the eyepiece, allowing direct visualization during intubation. The other version has a "Direct Coupled Interface" that displays the image directly on a video monitor. The eyepiece can also be converted to project the image onto a remote monitor to provide better visualization and enhance teaching. There is an adaptor "slide cone" for fixation of the ETT. This adaptor has a side port that allows oxygen insufflation.

Method of insertion: The ETT is loaded over the Bonfils optical stylet and fixed in such a way (keeping the tip of the

stylet at the ETT bevel) that a crescent of illumination is seen at the right side of the screen (**Fig. 9B**). Bonfils fiberscope is held like a pen and introduced through right angle of the mouth. The Bonfils fiberscope is advanced through the retromolar space and then turned cephalad to visualize the glottis. Preloaded ETT is then advanced into the trachea and the Bonfils fiberscope is withdrawn by rotating it toward foot-end of the patient.

Advantages: It is especially useful when the patient has expensive/delicate dental work, inconveniently placed solitary tooth, loose teeth, and large gaps between upper teeth which can trap laryngoscope blade and impede its movement. The external diameter of the adult Bonfils scope allows the use of ETTs as small as the 5-mm microlaryngeal tube and DLTs.

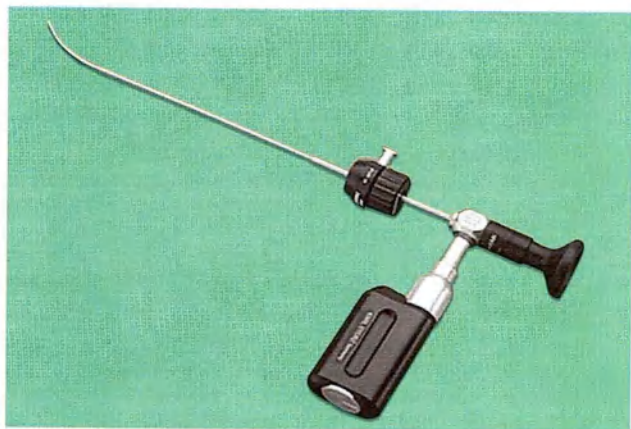


Fig. 9A: Bonfils retromolar intubating fiberscope.

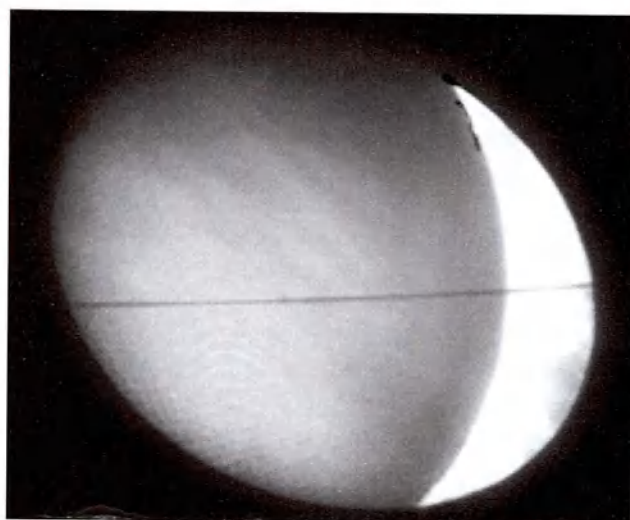


Fig. 9B: Crescent.

Disadvantages: Learning curve for its use is very high. It cannot be used for nasal intubations. The rigidity of

the scope may increase the risk of airway trauma in inexperienced hands. The nonmalleable shaft may limit the ability to angle the scope in cases where the larynx is extremely anterior.

Q. Describe King Vision videolaryngoscope.

Ans. King Vision videolaryngoscope (**Fig. 10**) is a portable battery-operated videolaryngoscope, which consists of two components, and has a usable display and a disposable blade. The display is LCD and 6.1 cm screen. Works with three AAA batteries which has a life of 90-minute usage.

Presently, only size 3 blade is manufactured as both standard and channeled types. The lens at the distal tip of the blade is coated with antifog material. Width (AP diameter) of the blade is 13 mm for standard blade and 18 mm for the channeled blade. The blades are 17 cm long and the channeled blade can accommodate ETT sizes 6.0–8.0 internal diameter. Stylet in ETT is not required while using the standard blade.

An optional video-out cable for connection to an external monitor is also available.

The limitations of King Vision present design are that it is available only in adult size 3, and it requires mouth opening of 2 cm if the channeled blade needs to be used.

Disinfection

After the use, disconnect the blade from display. Blades are disposable. Wipe blood and secretions on the display. Wipe the display using Sani-Cloth wipes (isopropyl alcohol) for 10 minutes. Change the wipe every 2 minutes during disinfection. Display is not submersible into any disinfectant solution. Let the scope air dry. Display materials are also compatible with Cidex, Cidex Plus®, or Cidex OPA or 70% isopropyl alcohol. Autoclave and ethylene oxide sterilization is not recommended.



Fig. 10: King Vision videolaryngoscope.

Q. What is Kepler intubation system (KIS)?

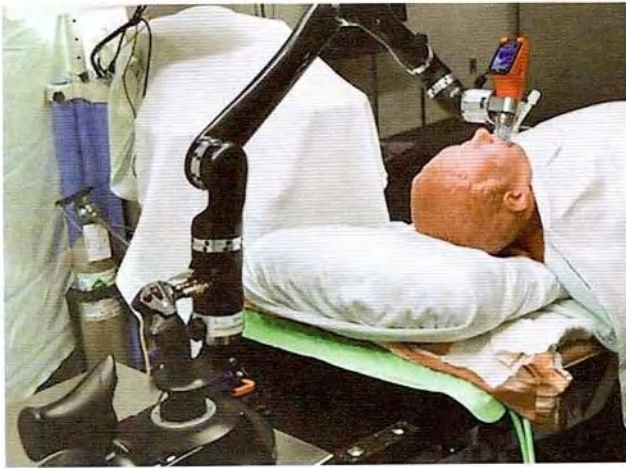


Fig. 11: Kepler intubation system.

Ans. It is a robotic intubation system, remotely controlling a standard videolaryngoscope with the option of semiautomated intubation or Pentax channeled scope mounted over the robotic arm. This system is named in honor of an astronomer, Johannes Kepler, best known for his laws of planetary motion. More research and development is awaited in this technique (**Fig. 11**).

SUGGESTED READING

1. Ahmed-Nusrath A. Video laryngoscopy. *Curr Anae Critical Care*. 2010;21:199-205.
2. American Society of Anesthesiologists Task Force on Management of the Difficult Airway. Practice guidelines for management of the difficult airway. An updated report by the American Society of Anesthesiologists Task Force on Management of the Difficult Airway. *Anesthesiology*. 2003;98:1269-77.
3. Arulkumaran N, Lowe J, Ions R, Mendoza M, Bennett V, Dunser MW. Videolaryngoscopy versus direct laryngoscopy for emergency orotracheal intubation outside the operating room: a systematic review and meta-analysis. *Br J Anaesth*. 2018;120(4):712-24.
4. Asai T. Videolaryngoscopes: Do they truly have roles in difficult airways? *Anesthesiology*. 2012;116(3):515-7.
5. Hemmierling TM, Wehbe M, Zaouter C, Taddei R, Morse J. The Kepler Intubation System. *Anaesth Analg*. 2012;114(3):590-4.
6. Holm-Knudsen R. The difficult pediatric airway—a review of new devices for indirect laryngoscopy in children younger than two years of age. *Paediatr Anaesth*. 2011;21(2):98-103.
7. Jaber S, De Jong A, Pelosi P, Cabrini L, Reignier J, Lascarrou JB. Video laryngoscopy in critically ill patients. *Critical Care*. 2019;23(1):221.
8. Jiang J, Ma D, Li B, Yue Y, Xue F. Video laryngoscopy does not improve the intubation outcomes in emergency and critical patients—a systematic review and meta-analysis of randomized controlled trials. *Critical Care*. 2017;21(1):288.
9. Karalapillai D, Darvall J, Mandeville J, Ellard L, Graham J, Weinberg L. A review of video laryngoscopes relevant to the intensive care unit. *Indian J Crit Care Med*. 2014;18(7):442-52.
10. Lee YK, Chen CC, Wang TL, Lin KJ, Su YC. Comparison of video and direct laryngoscope for tracheal intubation in emergency settings: A meta-analysis. *Acute Med*. 2012;2(2):43-9.
11. Myatra SN, Doctor JR. Use of video laryngoscopy as a teaching tool for novices performing tracheal intubation results in greater first pass success in neonates and infants. *Indian J Anaesthesia*. 2019;63(10):781-3.
12. Niforopoulou P, Pantazopoulos I, Demestiha T, Koudouna E, Xanthos T. Video-laryngoscopes in the adult airway management: a topical review of the literature. *Acta Anaesthesiol Scand*. 2010;54(9):1050-61.
13. Sun Y, Lu Y, Huang Y, Jiang H. Pediatric video laryngoscope versus direct laryngoscope: a meta-analysis of randomized controlled trials. *Pediatric Anesthesia*. 2014;24(10):1056-65.
14. Theodore Gar-Ling Wong. The bonfils retromolar intubation fibroscope: Advantages and practical aspects of its use. *AJA-Online.com*. 2011;12:40-8.
15. Xue FS, Li HX, Liu YY, Yang GZ. Current evidence for the use of C-MAC videolaryngoscope in adult airway management: a review of the literature. *Ther Clin Risk Manag*. 2017;13:831-41.

Cardiopulmonary Exercise Test (CPET/CPEX/CPX)

Vandana Agarwal, Vijaya Prakash Patil

Q. What is cardiopulmonary exercise testing (CPET/CPEX/CPX)?

Ans. Cardiopulmonary exercise test is an integrated global assessment of pulmonary, cardiovascular, hematological and musculoskeletal system which may not be reflected adequately when individual organ systems are assessed. Test evaluating resting functions of the heart and lung are not reliable predictors of perioperative risk. CPET on the contrary is a dynamic global assessment at rest and under stress thus indicating the physiological reserve of an individual. It is an objective evaluation of functional capacity and impairment of bodily systems. It is used for evaluation of undiagnosed exercise intolerance and exercise-related symptoms and also to categorize perioperative risks. The information derived from this test can be used to make informed decisions regarding optimal perioperative care of patient and also guide preoperative exercise prescription (when time permits) thereby improve patient's physical fitness prior to surgery.

Q. Briefly explain exercise physiology and the rationale for CPET.

Ans.

Exercise Physiology

During exercise, the metabolic demand increases progressively depending on the workload. To match this demand, the cardiovascular, pulmonary, and metabolic

systems have to increase their output. If there is mismatch between demand and supply, it leads to exercise limitation.

Oxygen (O_2) is taken into the body during inspiration by the lungs. It binds to hemoglobin and is distributed to the muscles/tissues by the circulatory system. At the cellular level, it is utilized for production of adenosine triphosphate (ATP) via aerobic pathways. Carbon dioxide (CO_2), a waste product of metabolism is transported in the opposite direction and excreted out of the body via the lungs. Exercise increases the energy demand, which in turn leads to increase in ventilation, cardiac output, and O_2 utilization (**Fig. 1**). Energy for muscle contraction comes from:

- Anaerobic hydrolysis of creatine phosphate. Creatine phosphate is high energy phosphate stored in the muscles available immediately for activity. It undergoes hydrolysis by creatine kinase to form creatinine and produces ATP. It provides large quantity of energy in the first few seconds of exercise before other metabolic procedures are activated, e.g., sprinting.
- Aerobic oxidation of glycogen and fatty acids occurs via tricarboxylic acid (TCA) cycle. Glycogen undergoes glycolysis to pyruvate which enters the Krebs cycle to produce ATP, CO_2 , and H_2O . Complete breakdown of one molecule of glycogen through the electron transport chain yields 39 ATPs. This mechanism sustains moderate-intensity exercise. Fatty acid undergoes β -oxidation to enter Krebs cycle. The rate of ATP

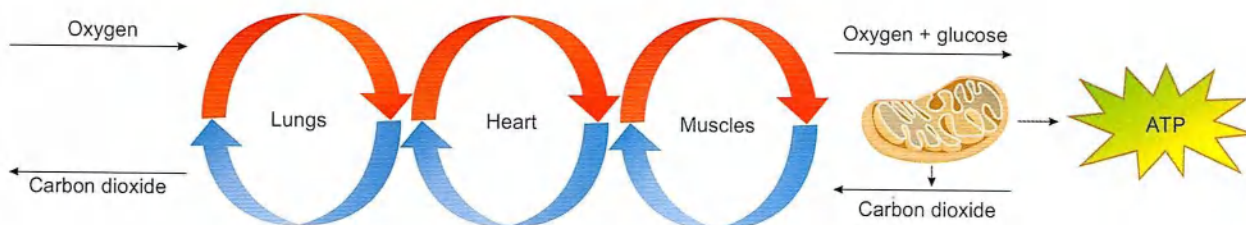


Fig. 1: Oxygen transport and utilization.

resynthesis from fat is too slow to be of great importance during high-intensity activity. At rest, fat is the preferred substrate and dominates the energy contribution. During exercise, carbohydrates are preferred when energy requirements increase at a rapid rate.

- Anaerobic oxidation of glycogen forms lactate via pyruvate. Aerobic oxidation continues until there is O_2 . In the absence of O_2 , pyruvate does not enter the Krebs cycle instead gets converted to lactate. Conversion of glycogen to lactic acid yields only 3 ATPs and the maximum rate of glycolysis can be reached within a few seconds of the onset of exercise. This mechanism comes into play during high-intensity exercise. Thus, supplementing the ATP formed by oxidative phosphorylation.

Rationale for CPET

Surgery is a catabolic state associated with increased O_2 demand by tissues. During the postoperative period, predominantly skeletal muscle protein is catabolized to provide amino acids along with lipolysis of triglycerides. During exercise, the O_2 supply and demand increase which must be matched by similar increase in ventilation and cardiac output to avoid failure of tissue perfusion. This information can be used to infer the patient's ability to perform metabolic work during the perioperative period assuming that the physiological response required to increase O_2 delivery is similar in both cases.

Gas exchange at the airway is used as a surrogate to assess chemical changes at the cellular level (with a few seconds lag). As the exercise intensity increases, there is a point when the aerobic metabolism cannot meet the demand this is when the anaerobic metabolism comes in to play and supplements the energy production. This is known as the anaerobic threshold, which can be determined by CPET, as the different metabolic pathways are being used for energy production during the exercise test.

Q. What equipment is needed for CPET?

Ans. A metabolic cart which consists of O_2 analyzer, CO_2 analyzer and flow meter is needed which measures the gas concentrations of O_2 and CO_2 and flow of air. In addition, a 12-lead electrocardiogram (ECG), continuous pulse oximetry, noninvasive blood pressure (BP) measuring device are required. All these are usually built in the system. Metabolic measurement device integrates the measurement of flow and gas concentrations to calculate the O_2 consumption (VO_2) and CO_2 production (VCO_2). Accurate alignment and calibration of flows and gas analysis is the key element of the CPET equipment. Faulty calibration can lead to significant source of error. Prior to conducting the test, all facilities available for resuscitation should be readily available.

Q. How is the test conducted?

Ans. Patient is consented and explained the need for the test. Information is given about how the test is done and what they should expect such as it gets harder to cycle as time passes and a minimum number of repetitions have to be maintained. Patient is made to sit comfortable on the bike (cycle ergometer) and is fitted with a mask and baseline observations and vital parameters recorded. Once the patient is comfortable and understands what is expected of him the test is initiated as mentioned below in the question on ways to stress the cardiopulmonary system for CPET.

Q. Various techniques for evaluation of cardiopulmonary reserve in perioperative period and how CPET is different from other methods?

Ans. The New York Heart Association (NYHA) functional classification is still used often. Assessment of functional capacity is routinely done by asking patients: "How many flights of stairs can you climb or how much distance can you walk without feeling breathless?" However, patients have a variable recollection and most of the times they overestimate or underestimate their true functional capacity. Hence, objective measurements are usually better than subjective reports. An ideal method for such an evaluation should be noninvasive, with high benefit/cost ratio, and portable for use.

The 6-minute walk test (6MWT): It is a practical simple test that requires a long corridor but no exercise equipment or advanced training for technicians. This test measures the distance that a patient can quickly walk on a flat, hard surface in a period of 6 minutes. An average healthy human walks around 575–750 m in 6 minutes. It evaluates the global and integrated responses of all the systems involved during exercise, including the pulmonary and cardiovascular systems, systemic circulation, peripheral circulation, blood, neuromuscular units, and muscle metabolism. Most patients do not achieve maximal exercise capacity during the 6MWT as they choose their own intensity of exercise in their comfort zone. The 6MWT is used as a measure of functional status of patients and predictor of postoperative morbidity and mortality. However, one has to keep in mind that the 6MWT does not determine peak O_2 uptake, diagnose the cause of dyspnea on exertion, or evaluate the causes or mechanisms of exercise limitation.

The shuttle-walking test: It is similar to the 6MWT, but it uses an audio signal from a tape cassette to direct the walking pace of the patient back and forth on a 10 m course. The walking speed is increased every minute, and the test ends when the patient cannot reach the turnaround point within the required time. The exercise performed is similar to incremental treadmill test. This test has a better correlation with peak O_2 uptake than the 6MWT.

Cardiac stress test: Total cardiac reserve involves the following reserves—heart rate, diastole volume, systole volume, coronary, metabolic and plasma norepinephrine. The measurement and evaluation of cardiac reserve is an important parameter in patients undergoing major and supra-major surgeries. The ECG in treadmill test, contractility and new-onset regional wall motion abnormality in dobutamine stress ECHO or perfusion defects on stress thallium scan are the optimal methods for analyzing myocardial chronotropism, dromotropism, and inotropism but they fail to test pulmonary reserve.

Cardiopulmonary exercise testing: It is most reliable and objective test for evaluation of functional capacity. It provides a safe, repeatable, noninvasive, individual assessment of combined pulmonary, cardiac, metabolic, circulatory and musculoskeletal function. It quantifies the functional ability to respond to the increased metabolic demands of major surgery generating a patient-specific measure of risk. CPET can differentiate the cause of dyspnea and provides a powerful diagnostic and prognostic tool for a variety of medical disorders, including coronary artery disease, cardiac failure, and restrictive and obstructive lung disease.

Q. What are the ways in which a cardiopulmonary system can be stressed for CPET?

Ans. Cardiopulmonary system can be stressed by making a subject exercise on treadmill, cycle ergometer or arm crank ergometer.

Treadmill: It is often used for exercise stress test; however, it only identifies cardiovascular problems. It can be used for CPET with added metabolic cart. When used for CPET it overestimates the VO_2 by 5–10% in comparison to a cycle ergometer. Body weight has greater impact on treadmill than cycle ergometer. ECG and BP measurements are difficult to record when compared to cycle ergometer. Quantification of external work is less easy with treadmill.

Cycle ergometer: It is commonly used for CPET with Ramp protocol or progressive incremental exercise. It typically consists of 3 minutes of unloaded pedaling (without any added resistance), thereafter the load increases (resistance created for pedaling) every minute usually by 5–25 watts/minute (W/min) depending on the individual fitness level. The patient is asked to exercise till they cannot do anymore. This is followed by 3 minutes of unloaded pedaling before stopping the test. The total test duration is about 8–12 minutes. A fit patient may be fine with 25 W/min increment, whereas the unfit may need 5 W/min. The work increment per minute depends on the patient's physical activity and can also be calculated from a nomogram, which depends on height, weight, and age.

Arm crank ergometer: It is useful for patients who are unable to use the cycle, e.g., lower limb amputees. Peak VO_2 attainable is 70% of that attained by cycle in healthy subjects. Also the lactate threshold is lower. This is not suited for patients with respiratory problems as the arm movements interfere with the use of accessory muscles of respiration. ECG and BP monitoring can be a problem.

Q. What information do we get from this test?

Ans. Total work done during the test, VO_2 , CO_2 produced, anaerobic threshold, respiratory response to exercise, metabolic equivalents for O_2 and CO_2 , desaturation if any, hemodynamic response, ECG changes.

Oxygen consumption: During the test, it depends upon ability of lungs to take in O_2 , factors determining transport of O_2 (cardiac output, O_2 carrying capacity of blood, O_2 dissociation curve shifts, muscle blood flow) and the ability of the muscle to utilize O_2 . It also depends on the volume of exercising muscles, age, sex, weight, physical conditioning, patient's effort and genetic factors.

Peak VO_2 represents the maximum aerobic capacity attained during the test. VO_2 max is the highest VO_2 attainable by the subject despite increases in workload; anything less is the peak VO_2 . It suggests that either stroke volume, heart rate, or tissue O_2 extraction has peaked.

It is reproducible in an individual unless there are pathophysiological changes. There is a linear relationship between the work done and VO_2 and is usually between 8.5–11 mL/min/W. A lower value indicates a problem with lungs, heart, and blood. A reduction of >50–60% of predicted VO_2 indicates high perioperative risk.

Anaerobic threshold: It characterizes the upper limit of aerobic metabolism during exercise as the intensity increases from to moderate to high work rate. If an individual continues to exercise, anaerobic metabolism is activated and supplements the aerobic metabolism. The point where this occurs is called anaerobic threshold. Fibers that rely on glycolytic pathway are recruited and hence, there is an outpouring of lactate and contributes to the increased CO_2 production. In normal subjects, anaerobic threshold occurs at about 50–60% of VO_2 max (range 40–80%) with training anaerobic threshold and VO_2 max can be increased. If anaerobic threshold is <10–11 mL/kg/min, it indicates high perioperative risk.

Cardiovascular system: One can assess the heart rate response to exercise and O_2 pulse, which is O_2 delivered and extracted every beat. Anemia, high carboxyhemoglobin (COHb), and hypoxemia influence it. Initial increase in O_2 pulse is due to increase in stroke volume, later on increased O_2 extraction contributes to its increase. If the stroke volume is limited, then O_2 pulse reaches its maximal value at

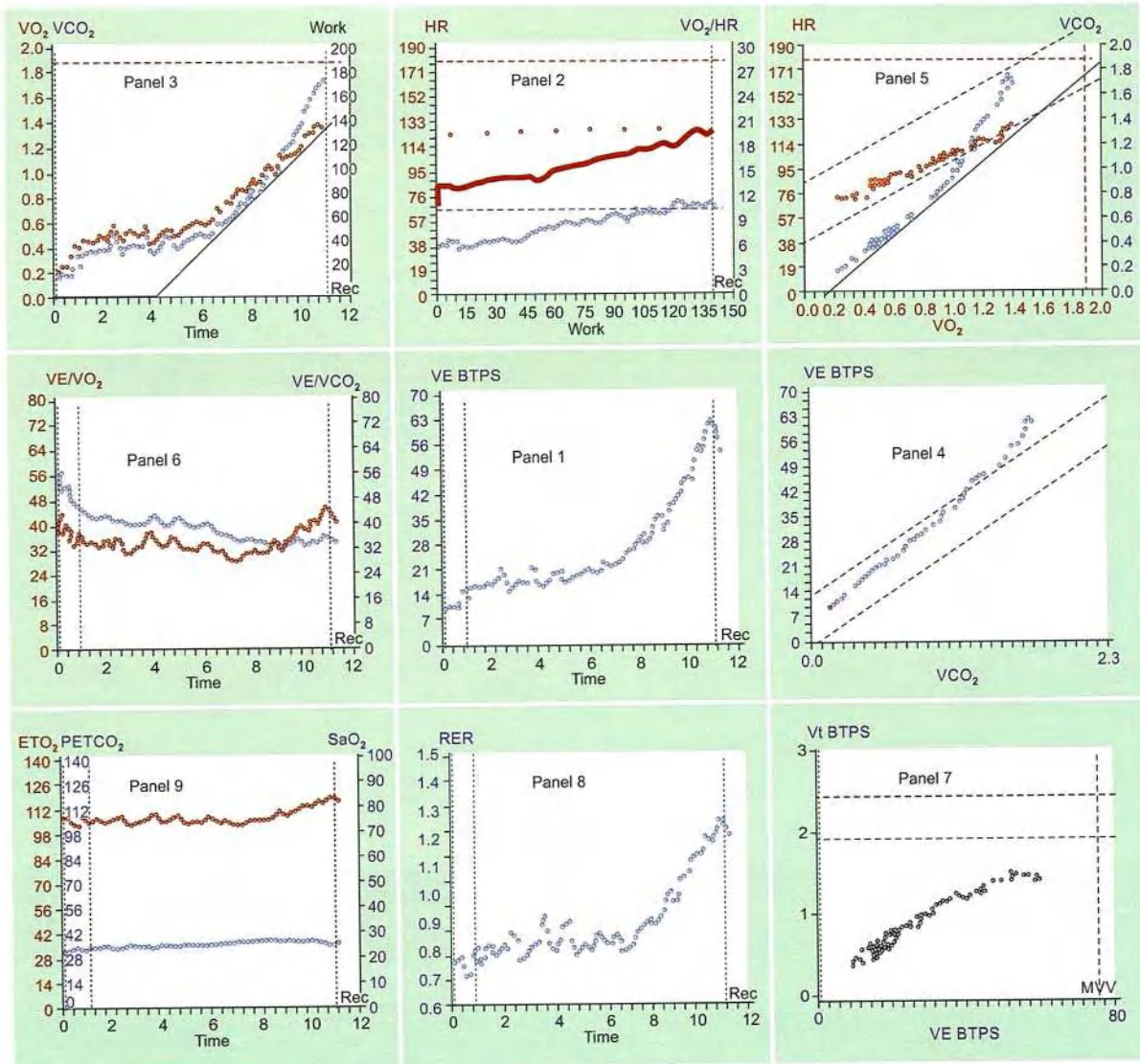


Fig. 2: Nine panel plot. Panels 3, 6, and 9 are used for deriving the anaerobic threshold. Panels 2 and 5 give information about heart rate and oxygen pulse. Panels 1, 4, and 7 give information about respiratory function. Panel 8 gives information of respiratory exchange ratio (RER) about the quality of test.

relatively low work rates resulting in early plateau indicative of heart disease.

Respiratory system: Respiratory pattern, rate, and minute ventilation (VE) give information about a patient's ventilator reserve. In early exercise, tidal volume (Vt) increases first with relatively stable respiratory rate (RR). As exercise intensity increases, parameters increase up to 70–80% of peak exercise, thereafter Vt stabilizes and further increase in VE is achieved by increase in RR.

Minute ventilation usually approaches 70% of maximal voluntary ventilation (MVV). The difference between MVV and maximum VE is the breathing reserve (BR). A lack of BR may suggest respiratory limitation of exercise. BR is usually more than 15% of MVV. For a given demand; patients with

chronic obstructive pulmonary disease (COPD), interstitial lung disease and pulmonary vascular disease may need a higher VE. This may be either because of high dead space, hypoxemia and ventilation-perfusion (V/Q) abnormalities.

In healthy subjects, lungs work efficiently as exercise increases, the ventilatory equivalents decrease with load. A higher ventilatory equivalent value indicates high dead space ratio or hyperventilation. In patients with respiratory diseases, the ventilatory efficiency is low, i.e., they have increase VE/ $\dot{V}CO_2$ and VE/ $\dot{V}O_2$ equivalents. A fall in saturation during exercise of >4% is considered significant for respiratory risk, similarly VE/ $\dot{V}CO_2$ >35 at the anaerobic threshold.

The above described information is extracted from a nine panel plot as shown in **Figure 2**.

Q. What are the contraindications for preoperative CPET?

Ans. **Table 1** shows the contraindications for preoperative CPET.

Q. What is the clinical utility of CPET?

Ans. Cardiopulmonary exercise testing has been evaluated in patients undergoing various surgeries. Anaerobic threshold, VO_2 peak, and ventilatory efficiency have been shown to be associated with outcomes as mentioned in **Table 2**.

Cardiopulmonary exercise testing is an excellent tool in our armamentarium to prognosticate patients undergoing major surgeries. It can be used preoperatively for risk stratification of patients undergoing major surgery, making informed decisions regarding the need for surgery and perioperative optimization and subsequent level of care. It is an objective assessment which can be used for preoperative exercise prescription for prehabilitation and assessment of patient's compliance with prehabilitation.

TABLE 1: Contraindications for preoperative cardiopulmonary exercise testing (CPET).

Absolute	Relative
Cardiovascular system: • Acute myocardial infarction • Unstable angina • Uncontrolled arrhythmias • Syncope • Uncontrolled heart failure • Acute endocarditis, myocarditis, pericarditis • Suspected dissecting aneurysm Respiratory system: • Uncontrolled asthma • Respiratory failure • Room air saturation of <85% Others: • Neurological deficit • Mental disorder	Cardiovascular system: • Left main stenosis • Moderate stenotic valvular disease • Untreated hypertension at rest (200/120 mm Hg) • Tachy- /bradyarrhythmias • Hypertrophic cardiomyopathy Others: • Electrolyte abnormalities • Pulmonary hypertension—significant • Pregnancy

TABLE 2: Cardiopulmonary exercise test variables and postoperative outcomes in various surgeries.

Surgery	No. of patients	Anaerobic threshold (AT) mL/min/kg	VO_2 peak or VE/VCO_2	Outcomes
Esophageal cancer ¹		<9.9	-	Significant increase in cardiopulmonary complications compared to AT > 11
Radical cystectomy ileal conduit ²	82	<12	-	Independent predictor of major postoperative complications and increased length of stay
Elective colorectal ³	196	<9.9	-	Associated with postoperative complications
Hepatectomy for hepatocellular carcinoma ⁴	61	<11.5	-	Independent prognostic indicator of both event-free survival and maintenance of Child-Pugh class
Hepatectomy ⁵	108	<9.9	$\text{VE}/\text{VCO}_2 > 34.5$	AT < 9.9 was 100% sensitive and 76% specific for in-hospital mortality, with a positive predictive value of 19% and a negative predictive value 100% Age and VE/VCO_2 at AT were significant predictors of postoperative complications
Major lung resection ⁶	225		$\text{VE}/\text{VCO}_2 > 35$	Strongest predictor of respiratory complications
Abdominal aortic aneurysm repair ⁷	415	<10.2	VO_2 peak < 15	AT was associated with 30-day mortality and ventilator efficiency was associated with 90-day mortality

REFERENCES

1. Moyes LH, McCaffer CJ, Carter RC, Fullarton GM, Mackay CK, Forshaw MJ. Cardiopulmonary exercise testing as a predictor of complications in oesophagogastric cancer surgery. *Ann R Coll Surg Engl.* 2013;95:125-30.
2. Prentis JM, Trenell MI, Vasdev N, French R, Dines G, Thorpe A, et al. Impaired cardiopulmonary reserve in an elderly population is related to postoperative morbidity and length of hospital stay after radical cystectomy. *BJU Int.* 2013;112(2):E13-9.

3. Lai CW, Minto G, Challand CP, Hosie KB, Sneyd JR, Creanor S, et al. Patients' inability to perform a preoperative cardiopulmonary exercise test or demonstrate an anaerobic threshold is associated with inferior outcomes after major colorectal surgery. *Br J Anaesth.* 2013;111(4):607-11.
4. West MA, Lythgoe D, Barben CP, Noble L, Kemp GJ, Jack S, et al. Cardiopulmonary exercise variables are associated with postoperative morbidity after major colonic surgery: a prospective blinded observational study. *Br J Anaesth.* 2014;112(4): 665-71.
5. Junejo MA, Mason JM, Sheen AJ, Moore J, Foster P, Atkinson D, et al. Cardiopulmonary exercise testing for preoperative risk assessment before hepatic resection. *Br J Surg.* 2012;99:1097-104.
6. Brunelli A, Belardinelli R, Pompili C, Xiumé F, Refai M, Salati M, et al. Minute ventilation-to-carbon dioxide output (VE/VCO₂) slope is the strongest predictor of respiratory complications and death after pulmonary resection. *Ann Thorac Surg.* 2012;93:1802-6.
7. Hartley RA, Pichel AC, Grant SW, Hickey GL, Lancaster PS, Wisely NA, et al. Preoperative cardiopulmonary exercise testing and risk of early mortality following abdominal aortic aneurysm repair. *Br J Surg.* 2012;99(11):1539-46.

Index

Page numbers followed by *f* refer to figure, *fc* refer to flowchart, and *t* refer to table.

A

- Abdominal binders 112
- Abdominal pain 149, 283
- Abdominal surgery
 - lower 276
 - major 368
- Abdominal trauma 144
- Abducens palsy 153
- Abruptio placentae 129
- Abscess 151
- Acceleromyography, principle of 561
- Accessory nerve 562
- Accidental dural tap, manage 112
- Accidental extubation 311
- Acetaminophen 55, 258, 335
- Acetazolamide 154, 156, 249
- Acetoacetic acid 322
- Acetone 322
- Acetylcholine 114, 216
- Acetylcholinesterase 107
- Achondroplastic disorders 152
- Acid 360
 - aspiration pneumonitis 131
- Acid-base 382
 - disorder, mixed 382
 - disorder 378, 379, 382
 - disturbance 146
 - homeostasis 376
- Acidemia 128
- Acidosis 8, 49, 63, 333
 - symptoms of 322
- Acquired atrioventricular block 58
- Acquired cardiovascular diseases 14
- Acquired causes 118
- Acrylonitrile 200
- Acute confusional state 241
- Acute coronary syndrome 28, 512
 - management of 521
 - treatment of 522*fc*
- Acute exacerbation, classification of 97*t*
- Acute kidney injury 342, 368
 - network 344
- Acute lung injury, transfusion-related 146
- Acute respiratory distress syndrome,
 - management of 393
- Adams valve 415
- Adenoid 346
 - facies 347
- Adenoidectomy
 - contraindications for 346
 - indications for 346
- Adenosine 10
 - triphosphate 200, 592
- Adequate preoxygenation 122
- Adjustable pressure-limiting 422, 453
 - valves 422, 451
- Administer muscle relaxant 465
- Adrenaline 262
- Adrenergic inhibitors 43
- Adrenergic receptor blockers 40
- Adrenocorticotrophic hormone 322, 368
- Adult chain of survival 481
- Advanced airway 478
 - placement of 478
- Advanced assessment tests 175
- Advanced cardiac life support 117, 476
- Advanced cardiovascular life support 514
- Advanced cerebral monitoring 172
- Advanced life support 487
- Advanced trauma life support 165
- Adverse cardiac events 19
- Adverse cardiovascular system 35
- Adverse drug interaction 220
- Aerosol face mask 570
- Afferent reservoir systems 454
- Agent-specific filling systems 437
- Agranulocytosis 304
- Air 400
- Airtraq optical laryngoscope 587, 588*f*
- Airway 168, 485, 489
 - abnormal 195
 - and breathing 117, 141
 - and control of cervical spine 196
 - and ventilation 477
 - assessment 162
 - axes, alignment of 177*f*
 - breathing, and circulation 195
 - cart, contents of 178
 - classified 489
 - compromise, worsening of 306
 - device placement 175
 - difficult 178
 - diseases 4
 - edema 306
 - exchange catheter 190
 - in mediastinal tumors, management of 184
 - injury
 - above larynx 194
 - below larynx 194
 - insertion of 493
 - involvement 178
 - history of 251
 - maintenance 165
 - management 133*fc*, 136, 171, 257
 - complicating 179
 - managing difficult 174
 - modified 489
 - obstruction 183, 311, 468, 493
 - causes of postoperative 212
 - complete 194
 - pediatric difficult 584
 - pressure release ventilation 393
 - resistance 183
 - sterilized 493
 - trauma, severe 465
 - types of 491*f*
 - uses of 489
- Albuminuria 37
- Alcohol
 - adverse effects of 268, 268*t*
 - consumption, excessive 136
 - intoxication, acute 275
 - withdrawal syndrome 275
- Alcoholic ketoacidosis 275
- Alfentanil 220, 258, 260, 336
- Alkalemia 383
- Alkali-free absorbents 424
- Alkalosis 63
- Allen's test 377
 - modified 377
- Allergic manifestations 113
- Allergic reactions 121
- Allodynia 204
- Alopecia 273
- Alpha-agonists 115, 248
- Alprazolam 39
- Alveolar hypoventilation 98
- Alveolar-arterial
 - gradient 216
 - oxygen gradient 216
- Alveolar ventilation 375
- Alzheimer's disease 153, 216
- Ambu AuraGain 541*f*
- Ambu bag 578*t*
- Ambu E valve 500*f*, 501
- Ambu Hesse valve 501
- Ambu laryngeal mask 540, 541, 541*f*
- Ambu resuscitation valve 498
- Ambu valve 500*f*
 - exhalation 500*f*
 - inhalation 500*f*
- Ambu-Hesse
 - expiration, working of 501*f*
 - inspiration, working of 501*f*
- Ambulatory epidural 111
- Amiodarone 8, 10, 262, 396
- Amitriptyline 204
- Amlodipine 116
- Ammonia 195
- Amniotic fluid embolism 129, 140-142
 - clinical features of 141, 141*t*
 - pathophysiology of 140
 - risk factors for 140
- Amphetamines 42
- Amygdalin 200
- Amyloid neuropathy 316
- Amyloidosis 179
- Anaerobic threshold 594
- Analgesia 171, 241
 - modes of 355
 - patient-controlled 202, 354, 355
 - perioperative 238
 - plan, postoperative 309
 - postoperative 73, 89, 158, 204, 223, 279, 299
- Anaphylactoid syndrome of pregnancy 140
- Anaphylaxis 178
- Anatomical variations 136
- Ancillary management 162
- Anemia 119, 120, 221, 333
 - causes of concern in 119
 - compensatory mechanisms in 119
 - in cirrhotic, causes of 274
 - in pregnancy 120
 - incidence of 119
 - of pregnancy, causes of 118
 - physiological changes and 118
 - prevent 119
 - risk factors for 120
 - severity of 119
 - treatment of 294
- Anemic patient, transfusion trigger in 122
- Anemic pregnant patient, anesthesia in 122
- Anencephaly 161
- Anesthesia 170, 222, 226, 321
 - breathing systems 451
 - care 248
 - compressed gases in 407
 - conduct of 49, 341
 - drugs 365*f*
 - epidural 129
 - equipment 373

- gas
 - cylinders 415
 - machine 415
 - induction of 56, 85, 95, 201, 211, 337, 341
 - inhalational 182
 - level of 549*t*
 - lighter planes of 8
 - local infiltration 338
 - low flow 426
 - machine 406, 427, 441
 - parts of 406
 - maintain 8
 - maintenance of 57, 80, 138, 170, 202, 211, 337
 - management 232
 - pharmacodynamics 360
 - pharmacokinetic principles in 360
 - practical applications 366
 - spinal 129
 - spontaneous 182
 - subarachnoid 238
 - technique of 89
 - type of 222
 - ventilators 386
- Anesthetic agents 204, 260*t*
 - effects of 298*t*
- Anesthetic causes 140
- Anesthetic considerations 9, 50, 52
- Anesthetic drugs 127, 306
- Anesthetic implication 421
- Anesthetic machine 420
- Anesthetic management, principles of 7
- Anesthetic technique 65, 79, 131, 137, 238, 297
 - type of 288
- Anesthetize 39
- Aneurysm 151
 - repair surgery 298
- Angina 18
 - pectoris, classification of 19
 - unstable 18, 42
- Angiotensin
 - infusion test 106
 - receptor blocker 23, 35
- Angiotensin-converting enzyme 35, 38
 - inhibitors 23, 218
- Animal tissues 9
- Anion gap acidosis
 - decreased 380
 - normal 380
- Ankle block 549*t*
- Ankle-brachial pressure index 71
- Ankylosing spondylitis 179
- Antacid prophylaxis 256
- Antecubital fossa 290
- Antepartum hemorrhage, complications with 146
- Anteroseptal myocardial infarction 520*f*
- Antiarrhythmia drugs 55
- Antibiotic 241
 - prophylaxis 7, 13
 - preoperative 256
- Anticholinergics 127
- Anticholinesterases 335
- Anticipate 114
 - difficult airway 483
 - massive obstetric hemorrhage 146
- Anticoagulant 33, 171, 265
 - drug 295
 - during pregnancy 13
 - management of 244
- Anticoagulation
 - indications for 9
 - therapy 551
- Anticonvulsant 156, 204
- Antidiabetic medications 238
- Antidiabetic treatment 317
- Antidiuretic hormone 282
- Antiemetic stockings 224
- Antifibrinolytics-tranexamic acid 150
- Antiglaucoma 248
- Antihypertensive agents 116
- Antihypertensive drug 38*t*, 40
 - therapy 107
- Antinuclear antibody 71
- Antiphospholipid antibody 71
- Antiplatelet 72
 - agents 33
 - drug 295
 - management of 244
 - therapy 13
- Antiproliferative agents 341
- Antipsychotics agents 322
- Antisymphotonic agents 248
- Antitachyarrhythmia function 63
- Antitachycardia pacing 62
- Antithrombotic management 12
- Antithyroid
 - antibodies 136
 - drugs 304, 305
 - side effects of 304
- Antler sign 5
- Anxiety, severe 42
- Anxiolytics 7
- Aorta 57
- Aortic dissection 42, 117
- Aortopulmonary shunt 47
- Apex posterior placement 64
- Apgar score 134
 - system 134, 134*t*
- Apical flow rumble 54
- Apixaban 235
- Aplastic anemia 118
- Apnea spells 53
- Apnea-hypopnea index 253
- Apneic oxygenation 187
 - during intubation, use of 122
 - limitations of 187
 - prerequisites of 187
 - techniques of 187
- Apparatus dead space 453, 453*f*
- Aqueduct stenosis 152
- Arachidonic acid metabolism 53
- Arachnoid cysts 151, 152
- Arachnoid granulations 152
- Arch of aorta, aneurysm of 465
- Arm crank ergometer 594
- Armored endotracheal tubes 468*f*
- Arndt blocker 471
- Arndt bronchial blocker 472*f*
- Arnold-Chiari syndrome 152, 163
- Arrhythmia 10, 84, 89, 223, 251, 275
 - by pacing, termination of 58
 - high risk for 205
 - significant 19
- Arterial blood
 - pressure 111
 - supply 504*t*
- Arterial blood gas 49, 87, 142, 194, 254, 270, 395
 - analysis 375
 - indications for 376
 - sample 377
 - error in 378
 - normal values in 379, 379*t*
 - physiological processes in 375
 - steps for analyzing 383
 - validation of 383
- Arterial cannula 306
- Arterial hypoxemia 566
- Arterial puncture
 - complications of 377
 - contraindications for 376
 - procedure for 377
- Arteriovenous fistula 338
- Arteriovenous malformations 151
- Aryngoscopy 115
- Aschner-Dagnini reflex 248
- Ascites
 - complications of 272
 - in cirrhosis, causes of 272
 - managed 273
- Aspiration 486
 - occurs 130
 - pharmacoprophylaxis 129
 - prophylaxis 114, 137
 - risk of 157
- Aspirin 10, 72, 134, 237, 360
- Asthma 90, 91, 91*t*
- Astrocytoma 151
- Asynchronous mode 62, 65, 66
- Asynchronous pacing 59, 65
- Atelectasis 89
- Atelectrauma 381
- Aterilization 579*t*
- Atherosclerosis 70, 72, 215, 221
- Atosiban 138
- Atracurium 8, 202, 258, 260, 276, 335
- Atrial fibrillation 11, 55, 214, 275, 517*f*
 - causes of 517
 - risk of 214
- Atrial filling pressure, increased 36
- Atrial flutter 55, 517*f*
- Atrial myxoma 5
- Atrial premature beat 22
- Atrial septal defect 52
- Atrial tachycardia 524*f*
- Atropine 157, 179, 249
- Auditory evoked potential 298
- Austin flint murmur 5
- Autoimmune causes 136
- Autologous donation, preoperative 295
- Automated external defibrillator 480, 487
- Automatic nonbreathing valve, types of 441
- Automatic tube compensation 394
- Autonomic nervous system 38, 214
- Autonomic neuropathy 314, 315, 317
- Auto-positive end-expiratory pressure, clinical
 - implications of 390
- Autoregulation 169
- Avogadro's hypothesis 431
- Awake intubation 189*f*
- Awake tracheal intubation 181
- Axillary block 559*t*
- Axillary brachial plexus block 290
- Axis of heart, calculation of 506*f*
- Axonal injuries, diffuse 172
- Ayre's T-piece 457, 457*f*, 460
- Azathioprine 341

B

- Back pressure valves 437
- Backward upward rightward pressure 497
- Bacteremia 232
- Bacterial infections 86
- Bag mask ventilation, difficult 175
- Bag refill valve 579
- Bag squeezers 386
- Bain's circuit 458, 458*f*
 - advantages 459
 - disadvantages of 459
 - functional analysis of 459*f*
- Balloon mitral valvotomy 8
- Barbiturate administration 172
- Barbiturate coma 171
- Barbiturate therapy, high-dose 173
- Bardex airway 493

- Bare-metal stent 28
 - Bariatric surgery 259
 - Barium lime 424
 - Barotrauma 391
 - manifestations of 391
 - Basic life support 486*t*
 - sequence 476
 - pediatric 487*f*
 - Baska mask 546, 546*f*, 547*f*
 - Bed rest 112
 - Benumof's 11 parameter analysis 175
 - Benzodiazepines 127, 220, 257, 276, 322
 - Benzyl isothiocyanate 200
 - Berman airway 490
 - Berman intubating airway 491
 - Bernoulli effect 252
 - Bernoulli principle 572
 - Beta-blockade therapy, perioperative 26
 - Beta-blockers 115
 - non-selective 249
 - Bicarbonate 324
 - Bicarbonate therapy
 - complications of 381
 - indications for 381
 - Bidirectional flow systems 455
 - Bier's block 288, 292
 - Bifascicular block 58
 - Biguanides 317
 - Bilevel inspiratory positive pressure ventilation 392
 - Biliary cirrhosis 267
 - Bilirubin formation 265
 - Bilirubin overproduction 279
 - Bimanual uterine compression 146
 - Binasal airway 493
 - Biochemical changes 216
 - Bioprosthetic heart valves 13
 - Bioprosthetic valves 9
 - Biosensors 55
 - Biosynthetic human-sequence insulins 318
 - Biotransformation 275
 - Biotrauma 391
 - Biotronik synchronous mode 62
 - Bipolar leads 59, 504
 - Bipolar pacing 60*f*
 - Bispectral index 222, 239
 - Bizzarri-Giuffrida blade 496
 - Bladder catheterization 166
 - Blades of Airtraq, types of 588*t*
 - Blalock-Taussig shunt 47
 - Bleomycin 396
 - Blind intubation techniques 183
 - Blind nasal
 - approach 182
 - intubation 180
 - Blockbuster laryngeal mask airway 548*f*
 - Blood 147, 152
 - and blood components, replacement of 296
 - calculate oxygen content of 378
 - cell transfusions 121
 - cross-match 237
 - flow
 - detection 71
 - reduced 217
 - glucose 197, 201
 - grouping 162, 201, 237
 - patch 112
 - products 147
 - supply to liver 264
 - transfusion, indications for 120
 - Blood loss 118, 143, 202, 231
 - causes of increased 231
 - degree of 143
 - during surgery 212
 - increased 217
 - Blood pressure 36, 104, 143, 157, 196, 324
 - diastolic 227
 - elevation of 35
 - low 143
 - noninvasive 49, 80, 237, 278, 465
 - systolic 35, 167, 227
 - Blood sugar
 - control 326, 328
 - drugs affect 322
 - management 319
 - perioperative 325
 - levels 237
 - managed postpartum 330
 - postprandial 320
 - preoperative control of 317
 - rise in 319
 - Blood-brain barrier 170, 364
 - B-lymphocyte 146
 - B-mode 6
 - Body
 - compartments 218
 - mass index 179, 250
 - plethysmography 399
 - surface area 191, 192, 198
 - calculation of 191*f*, 192*f*
 - Bolus hypertonic saline 173
 - Bone
 - cement implantation syndrome 240
 - tissues 527
 - Bonfils retromolar intubating fiberscope 589, 590*f*
 - Bovine 9
 - Boyle's bottle 438, 439
 - Boyle's law 431
 - Brachial plexus
 - block 289
 - techniques 290
 - Bradyarrhythmias 56
 - Bradycardia 128, 247
 - adult 515*f*
 - risk of 312
 - Bradykinin 193, 204
 - Brain 38, 69, 216
 - tissue oxygenation 172
 - Brain injury 168*t*
 - mild 171
 - moderate 171
 - primary 167, 168
 - secondary 168
 - severe 171
 - severity of 171
 - Brain trauma
 - foundation 166
 - guidelines, pediatric 172
 - Breath
 - assisted 387
 - controlled 386
 - dual control within 393
 - shortness of 42, 113, 382
 - types of 386
 - Breathing 165, 196, 259, 485
 - circuit 452
 - oxygen 382
 - Breathing system 451, 454*f*
 - combined 461
 - common components of 451
 - requirements of 451
 - Brimonidine 248
 - Bromage scale 549, 549*t*
 - modified 549*t*
 - Bronchial blocker 81, 82, 471
 - advantages of 473, 473*t*
 - disadvantages of 473
 - left-sided 82
 - types of 471
 - Bronchiectasis 86, 86*f*, 87, 530
 - clinical features of 86
 - etiology of 86
 - pathological changes in 86
 - predisposing factors for 86
 - with lung abscess 86
 - Bronchitis, chronic 90
 - Bronchodilator therapy 79
 - preoperative 87
 - Bronchomalacia 402
 - Bronchopleural fistula 89, 101
 - Bronchopulmonary dysplasia 53
 - Bronchospasm 72, 129
 - Brook airway 490, 491*f*
 - B-type natriuretic peptide 55, 71
 - Bubble-through vaporizer 435
 - Buck's fascia 351
 - Buerger's disease 69, 69*t*
 - Bulging fontanelle 158
 - Bulk oxygen storage 407
 - Bullard laryngoscope 195, 496, 587*f*
 - Bullard rigid fiberoptic laryngoscope 587
 - Bundle branch block 62
 - Bupivacaine 111, 127, 289, 292
 - S-enantiomer of 111
 - toxicity 113
 - Burden pressure gauge 414*f*
 - Burn 178, 191, 196
 - dressing, anesthesia for 202
 - drugs affected in 203
 - injury 193, 203, 204
 - major 201
 - location of 201
 - pain, mechanism of 204
 - prognosis with 192
 - severity of 191
 - shock 193
 - wounds 198
 - Burning charcoal 199
 - Bypass surgery 72
 - Bypass valve 416
 - Bypass vaporizer, variable 433, 433*f*
- C**
- Caffeine 112
 - Calcineurin inhibitors 341
 - Calcium 333
 - channel blockers 115, 138
 - gluconate 478
 - hydroxide 423
 - metabolism 106
 - Calibrate oxygen analyzer 428
 - Canadian C-spine rule 168
 - Canagliflozin 318
 - Cancers 183
 - Cannula cricthyrotomy 185
 - Captopril 46
 - Carbamazepine 172
 - Carbohydrate metabolism 264
 - Carbon dioxide 169, 410
 - partial pressure of 155
 - Carbon monoxide 79, 198, 199, 395, 425
 - lung for 401
 - Carbonic anhydrase inhibitor 154
 - Carboprost 141, 149
 - Carboxyhemoglobin 91
 - effects of 93
 - Cardiac arrest 205, 476, 479
 - causes of 478*t*, 485, 486
 - emergency cesarean delivery in 483
 - manage 142, 480*f*
 - mechanism of 486
 - rhythms 486
 - victim after 479

- Cardiac arrhythmias 52
- Cardiac cachexia 48
- Cardiac catheterization 48, 55
- Cardiac complications 314
- Cardiac defects 210
- Cardiac disease 117, 282
- Cardiac dysrhythmias 205
- Cardiac events 71
- Cardiac function 50
- Cardiac ischemia 71
- Cardiac myoglobin 199
- Cardiac output 83
 - minimize reduction in 122
 - monitoring 239
- Cardiac resynchronization therapy 61
- Cardiac rhythm management device 61
- Cardiac risk assessment
 - methods of preoperative 23
 - perioperative 23
- Cardiac risk indices 22*t*
- Cardiac status, evaluation of 61
- Cardiac stress test 594
- Cardiac surgery
 - adult 45
 - pediatric 45
- Cardiac troponins 32
- Cardiogenic pulmonary edema 141
- Cardiopulmonary 282
 - cerebral resuscitation 116, 134
 - evaluation of 593
 - exercise 77, 596
 - test 21, 79, 592, 594, 596*t*
 - function 287
 - system 594
- Cardiopulmonary bypass 11, 45
 - implications of 14
 - role of 184
- Cardiopulmonary resuscitation 196, 262, 476, 480, 487, 522
 - compression-only 484
- Cardiorespiratory
 - assessment, preoperative 78
 - compromise 228
- Cardiothoracic ratio 527*f*
- Cardiotocography, continuous 128
- Cardiovascular abnormalities 333
- Cardiovascular changes 122, 141, 297
- Cardiovascular collapse 84
- Cardiovascular complications, perioperative 26
- Cardiovascular disease 14, 402
 - risk of 35
- Cardiovascular homeostasis 38
- Cardiovascular manifestations 42
- Cardiovascular signs 113
- Cardiovascular stability 168
- Cardiovascular system 93, 156, 227, 229, 275, 302, 594
 - changes in 219
 - function 251
- Cardiovascular workup 77
- Cardioversion 481
- Carditis 5
- Carlens tube 469, 469*f*
- Carotid artery disease 74
- Carotid endarterectomy 298
- Carpenter's syndrome 53
- Carrier gas flow, velocity of 435
- Catalases 199
- Cataract 205, 244
 - congenital 245
 - surgery 244, 248, 327
- Catecholamine 109
- Catheter
 - ablation 58
 - indicated 80
- Cauda equina 160
- Caudal block 352
 - complications of 352
 - technique of 352
- Cell
 - membrane adenosine 193
 - salvage, intraoperative 147, 295
 - saver 143
- Cellular plasma fibronectin, increased 106
- Central cholinergic syndrome 220
- Central nervous system 156, 165, 209, 216, 226, 271, 299*t*, 302
 - diseases, age-related 216
 - examination 271
 - signs 113
- Central neuraxial blockade 258
- Central pontine myelinolysis 230
- Central retinal artery occlusion 247, 299*t*
- Central venous pressure 7, 49, 123, 197, 258
 - monitoring 239
- Cerebellar dysfunction, signs of 152
- Cerebral autoregulation 216
- Cerebral blood
 - flow 155, 169, 216
 - volume 155
- Cerebral hyperemia 169
- Cerebral hypoperfusion 170
- Cerebral infection, prevent 166
- Cerebral ischemia 39, 169
- Cerebral oxygen
 - consumption 216
 - saturation 239
- Cerebral perfusion
 - impaired 157
 - pressure 157, 168
- Cerebral pontine myelinolysis 170
- Cerebral vascular disease 315
- Cerebrospinal fluid 84, 126, 151, 155, 163, 166, 299
 - amount of 151
 - blockade 151
 - causes of 151
 - drainage 171, 172
 - volume of 217
- Cerebrovascular accidents 221
- Cerebrovascular disease, history of 254
- Cervical
 - and atlanto-occipital joint function,
 - assessment of 174
 - epidural anesthesia 312
 - plexus blocks, role of 312
 - spine 166, 533*f*
 - control 165
 - fracture 533*f*
 - injury 465
 - motion restriction 166
 - movement of 583
 - tear 143
 - vertebrae 130
- Cervix, condition of 108
- Cesarean section 12
 - advantage 12
 - disadvantages 12
 - previous 144
- Chandy's maneuver 537
- Charles's law 431
- Check valve assembly 413
- Chemical burns 205
- Chemical prophylaxis 224
- Chemical tracheobronchitis 194
- Chemical ventriculitis 152
- Chemotaxis 327
- Chemotherapy
 - preoperatively 75
 - related toxicity 75
- Chest
 - compression
 - create blood flow 484
 - technique of 485
 - drain
 - complications of 103
 - function of 99
 - insertion, indications for 99
 - drainage system, components of 99
 - pain 113, 141
 - physical therapy 403
 - radiography 54
 - tube
 - insertion of 102
 - insertion, procedure of 100
 - management of 102
 - sizes 100
- Chevalier Jackson position 496
- Chiari malformation 151, 152
- Child's heart and lungs, evaluation of 50
- Child-Pugh classification 273, 274
- Child-Turcotte-Pugh classification 273, 274*t*
- Chloride 468, 195
- Cholelithiasis 300
- Cholestasis 265
- Cholinergics 249
- Chondrodysplasia syndrome 245
- Chordae 5
- Choroid
 - plexectomy 154
 - plexus 151
 - papilloma 151
- Chromosomal inversions 136
- Chronaxy 554
- Chronic disease, anemia of 118, 294
- Chronic kidney disease, stages of 332
- Chronic medications, management of 23
- Chronic obstructive pulmonary disease,
 - hallmark of 90
- Chronic therapy, withdrawal of 41
- Chronotropic incompetence 61
- Chylothorax 84, 300
- Cigarette smoke 200
- Cilostazol 72
- Circle system 421, 422*f*
 - advantages of 422
 - disadvantages of 422
 - functional analysis of 426*f*
- Circuit gas delivery system 89
- Circuits by function, classification of 453*t*
- Cirrhosis 275
 - causes of 267
 - classified 274
 - complications of 268
 - etiologic classification 274
 - functional classification 274
 - histologic classification 274
 - morphologic classification 274
 - of liver 267, 274
 - with portal hypertension 264
- Cisatracurium 260, 335
- Claudication 69
- Clayton yellow 424
- Clean intermittent catheterization 164
- Clear fluids preoperatively 370
- Cleft lip 207, 207*f*, 208*t*
 - anesthesia for 207
 - types of 207
- Cleft palate 207, 207*f*, 208*t*
 - types of 207
- Clonidine 42, 115, 248
- Clopidogrel 72, 237
- Clotting studies 336
- Club feet 162

- C-mac videolaryngoscope D blade 586f
 Coagulation activation 106
 Coagulation disorders 143
 Coagulation system, changes in 125t
 Coagulopathy in cirrhosis 274
 Coarctation of aorta 37
 Coaxial circuits 459
 Cobra perilaryngeal airway 540, 543f
 Cocaine 42
 Cockcroft-Gault equation 338
 Cockcroft-Gout formula 217
 Codeine 134, 336
 Cognitive dysfunction, postoperative 73, 224, 241
 Cognitive functions 221
 Cohen flexitip blocker 471, 472f
 Cole's tube 466, 466f
 Collateral circulation, adequacy of 377
 Colles' fracture 288, 289
 features of 288
 management of 288
 Colloid cysts 151
 Color Doppler 54
 Color-coded knobs in flowmeter 420f
 Combined spinal epidural 110
 anesthesia 238
 Combitube 474, 474f
 Common tests, predictive ability of 176t
 Compartment syndrome 198
 development of 198
 Complete blood count 71, 75, 87, 197, 236, 323
 Complete heart block, diagnose 512
 Complex partial seizures 272
 Compressed gas 406
 Compression-relaxation ratio 477
 Compression-ventilation ratio 477
 Conception, before 13
 Conductive system 214
 Confusion 199
 Conjunctiva 246
 Conn's syndrome 37
 Connell airways 490
 Conservative management 148
 Context-sensitive half-time 365
 Continuous positive airway pressure 81, 89, 187, 256, 472
 Contralateral deviation 85
 Conventional fluid management 369
 Cook's modification 175
 Coopdech blocker 472
 Coopdech endobronchial blocker 472f
 Copper kettle 444, 444f
 classification 444
 Coracoid approach 290
 Cormack-Lehane
 classification grades 497t
 grading 175, 497, 497f
 Corneal abrasions 286
 Coronary anatomy 17
 Coronary angiography 20, 21
 Coronary artery
 bypass grafting 25, 28, 234
 disease 28, 39, 251, 314
 right-sided 17
 left main 17
 right 17f
 system 17f
 Coronary blood flow 18, 215
 normal 17
 Coronary computed tomographic angiography 23
 Coronary heart disease 314
 Coronary sinus 61
 Corpus callosum, atrophy of 153
 Cortical blindness 153
 Corticosteroid
 administration of 138
 high-dose 130
 Cough 103, 141
 test 92
 Cracking valve 412
 Cranial blood, evacuation of 170
 Cranial nerve
 injury 166
 palsy 153
 Crawford cannula 352
 Crawford point needle 551
 Cricoid pressure 130, 185
 trainer 130
 Cricothyrotomy
 emergency 133
 surgical 185
 Critical leg ischemia 71
 Critical limb ischemia 70
 Critical pressure 407
 Critical temperature 432
 Crystal 229
 Cuff 463
 on red rubber tubes 463
 oropharyngeal airway 492, 492f
 types of 464
 Cuffed perilaryngeal sealers 540
 Cuffed pharyngeal sealer 543
 Cuffed tube, advantages of 463
 Cuffless preshaped sealer 544
 Curling's ulcers 194
 Current output control 555
 Cushing's syndrome 36, 37
 Cushing's triad 158, 171
 Cutaneous nerve, posterior 291
 Cyanide 200
 poisoning 200
 causes of 200
 pathophysiology of 200
 Cyanosis 46, 48, 54, 56, 129
 Cyanotic congenital heart disease 210
 Cyanotic spells, effect of 50
 Cycle ergometer 594
 Cyclical shear stress injury 391
 Cyclooxygenase-2 553
 Cyclopentolate 249
 Cyclopropane 410
 Cyclosporine 341
 Cylinder 430
 contents of 408
 gauge 427
 made 407
 pressure gauge 414
 pressure of 409
 sizes of 407
 tested 409
 valve 410, 411
 open 412
 slowly 412
 Cystic fibrosis bronchiectasis 86
 Cytochromes 199
 Cytokines 368
 Cytomegalovirus 118
- D**
 Dalrymple sign 302
 Dalton's law 431
 Dandy-Walker syndrome 152
 Dapagliflozin 318
 Datex-Ohmeda link-25 system 418f
 Dead space ventilation 453
 Decompressive craniectomy 172
 Deep hypothermic cardiac arrest 45
 Deep venous thrombosis 97, 224, 238, 250
 prophylaxis 7
- Defibrillation 481
 biphasic 481
 monophasic 481
 work 481
 Degludec 319
 plus 319
 Dehydrates endothelial cells 170
 Dehydration, symptoms of 322
 Delirium 224, 225, 241
 treatment of 225
 Demand valve 577
 Dementia 153, 221
 Deoxygenated blood 57
 Deoxyribonucleic acid 168
 Dermal injury 199
 Dermatomal origin 112
 Dermis 192
 part of 191
 Desflurane 335, 425
 vaporizer, working of 447f
 Detemir 319
 Deterioration, signs of 171
 Detoxification 265
 Dexmedetomidine 47, 223, 225, 356
 use of 225
 Dextrose 170, 198
 Diabetes 214, 215
 complications of 314
 immune-mediated type 1 313
 poorly controlled 53
 type 1 313
 idiopathic 313
 type 2 313
 type of 314
 Diabetes mellitus 72, 136, 178, 221, 226, 313
 classification of 313
 drug-induced 313
 insulin therapy for 383
 symptoms of 251
 Diabetic amyotrophy 314
 Diabetic autonomic neuropathy 130, 314
 differential diagnosis of 316
 management of 317
 tests for 316
 Diabetic ketoacidosis 318, 323
 treatment of 323
 Diabetic mothers 330
 Diabetic nephropathy 314
 Diabetic neuropathy 314
 Diabetic patient, surgery in 319
 Diabetic retinopathy 314
 Diagnostic peritoneal lavage 166, 167
 Dialysis disequilibrium syndrome 334
 Diaphoresis 113
 Diaphragmatic hernia, congenital 530f
 Diarrhea 149
 Diastolic dysfunctions 214
 Diastolic murmur 5
 Diastolic relaxation, decreased 36
 Diastolic velocity 153
 Diazepam 39, 201, 220, 233, 292
 metabolites of 276
 Dietary habits 251
 Difficult airway
 cart 178f
 management 176
 trolley 178t
 Difficult surgical airway, assessment for 175
 Diffusion hypoxia 97
 Digitalis toxicity 523
 Digoxin 8, 10, 46
 Dihydrocodeine 336
 Dihydroxyphenylalanine 107
 Diltiazem 8

Direct acting valves 411*t*
 Direct cardiac effects 113
 Direct cutaneous injection 212
 Direct laryngoscopy 175, 183*f*, 581, 582*f*
 Disability 197
 Disconnection technique 473
 Diseases linked to obesity 250*t*
 Disinfection 579*t*, 585
 Disposable classic version 539*f*
 Disseminated intravascular coagulation 140, 199, 265
 Distal tracheomalacia 402
 Diuretic therapy 153
 Dizziness 149, 199
 Dobutamine stress
 echocardiography 21
 test 61
 Donor skin 201
 Dopamine 343, 360
 levels of 216
 Doppler echocardiography 6, 54
 Doppler ultrasound 71
 Doppler velocimetry studies 106
 Dorsal penile nerve block 351*f*
 Double lumen tube, correct positioning of 471
 Double-burst stimulation 563
 Double-lumen endotracheal tube 81, 81*t*
 advantages of 473
 complications of 471
 disadvantages of 473
 Double-lumen tube 81, 469, 470*f*
 advantages of 473*t*
 and bronchial blockers, disadvantages of 473*t*
 right 471*f*
 types of 469
 Down syndrome 208, 245
 Drug
 hepatotoxicity, mechanisms of 278
 history 251
 therapy 478
 toxicity 140
 treatment 35
 Drug-eluting stent 25, 28
 Dual antiplatelet therapy 28
 management of 28*f*
 Dual-chamber pacing 59
 Duckett-Jones criteria 5
 Ductus arteriosus, embryology of 53
 Ductus venosus 52
 Dural puncture epidural technique 132
 Dynamic compliance 392
 Dyslipidemia 72
 Dyspepsia 149
 Dyspnea 71, 85, 91, 103, 141

E

Ear
 abnormalities 209
 infection 158
 Echocardiogram 142, 211
 Echocardiographic systems 6
 Echocardiography 21
 principles of 6
 two-dimensional 6, 87
 Eclampsia 42, 105, 108
 Edema 252
 Efferent reservoir systems 455, 458
 Ehlers-Danlos syndrome 152
 Eisenmenger's syndrome 54
 Elastic elements, loss of 215
 Elbow, block of nerves at 290
 Elective noncardiac surgery 23, 27
 Electrical burns 205

Electrical nerve locator 555
 Electrocardiogram 138, 320
 Electrocardiography 404
 Electrocautery, effect of 65
 Electroconvulsive therapy 66
 pacemaker for 66
 Electrode placement 477
 Electrolytes 274
 Electromyography endotracheal tube 310*f*
 Electronic extubation 394
 Electronic flowmeters work 420
 Elisha airway device 545, 546*f*
 Embolism 486
 Embryology 208
 Emergency medical service 522
 Emergency oxygen supply 427
 Emergency surgery 304
 Empagliflozin 318
 Emphysema 530*f*
 Empyema 84, 89
 Enalapril 116
 Encephalitis 151
 Encephalocele 151, 161
 Enclosed afferent reservoir system 457, 457*f*
 Endarteritis 55
 Endobronchial tube 81
 Endocardial pacing 59
 Endocrine 125
 changes 106
 disturbances 334
 factors 136
 neoplasia, multiple 301
 system 218
 Endothelial cell activation 105
 Endotoxemia 194
 Endotracheal intubation 115, 157, 348
 complications of 468
 procedure of 465
 Endotracheal tube 181, 182, 462, 463*t*, 534
 cuff system of 463
 intolerance of 41
 selection of 466*t*
 tissue reactivity of 462
 types of 466
 End-stage liver disease model, pediatric 273
 End-tidal capnometry 49
 Enflurane 425
 Entonox 109, 410, 413
 cylinders 413
 separation of 413
 Environmental causes 136
 Ephedrine 305
 Epicardial pacing 60
 Epidermis 192
 completely destroyed 191
 Epidural and spinal drug spread 551
 Epidural anesthesia 115, 131, 138, 238, 550, 551
 Epidural blood patch, technique of 112
 Epidural catheter 8
 Epidural hematomas 172
 Epidural needles 551
 types of 550
 Epiglottitis 465
 Epistaxis 42
 Epstein-Barr virus 118
 Epstein-Macintosh-Oxford
 apparatus, arrangement of 442
 inhaler 439, 440*f*
 Equipment-related potential harm 569, 571, 574
 Ergometrine 149
 contraindications 149
 side effects 149
 Ergot alkaloids 115
 Erythema marginatum 5

Erythrocyte
 loss of 193
 sedimentation rate 124
 Erythroid cells 265
 Erythropoietin, perioperative 294
Escherichia coli 318
 Esmolol 8
 hydrochloride 43
 Esophageal obturator airway 491, 492*f*
 Esophageal tracheal
 combitube 540
 tube 478
 Ethanol 360, 365
 Ether
 anesthesia 438
 vaporizer 439
 Ethyl
 chloride 433
 orange 424
 violet 424
 Ethylene oxide 475
 Eurocollins 340
 Euthyroid sick syndrome 303
 Everolimus- and zotarolimus-eluting stents 25
 Evoked motor response 556*t*
 Evoked potential monitoring 298
 Ex utero intrapartum treatment 164
 Excessive dextrose 275
 Excitatory neurotransmitter 230
 Excitotoxicity 168
 Excretory function 265
 Exercise
 physiology 592
 stress testing 23
 tolerance 56, 251
 Exercise-induced bronchoconstriction test 402
 Exocrine pancreatic diseases 314
 Expiration diverter 577
 Exponential process 363
 Exposure and environment control 197
 Extracorporeal membrane oxygenation 142
 Extradural hematoma 169, 172
 Extrahepatic cholestasis 279
 Extrathoracic obstruction 402
 variable 402*f*
 Extraventricular drain 154
 Eye
 long 247
 movement, rapid 252
 nerve supply to 245
 signs 302
 Eyeball 245
 EZ-blocker 472, 472*f*

F

Fabry disease 3
 Face mask 203, 534, 574*f*
 ventilation 134
 Facial block 246
 Facial edema, extensive 181
 Facial nerve, blocking 246*f*
 Failure to pace 64*f*
 Fasting blood sugar 320
 Fastrach endotracheal tube 537
 Fat
 embolism syndrome 239
 diagnosis of 240*t*
 metabolism 264
 Fatigue 199
 Femoral artery, superficial 70
 Femoral nerve block 559*t*
 Femorofemoral cardiopulmonary 308
 bypass 308

- Fenoldopam 43, 343
 - Fentanyl 115, 127, 220, 260, 336, 360
 - Fetal alcohol syndrome 209
 - Fetal anesthesia, ensuring adequate 164
 - Fetal asphyxia 136
 - Fetal circulation 52, 53*f*
 - normal 52
 - Fetal complications 108, 146
 - Fetal distress 114
 - causes of 128
 - Fetal ductus arteriosus 139
 - Fetal heart rate
 - continuous 107
 - external 138
 - Fetal heart sounds 111
 - Fetal heartbeat 145*f*
 - Fetal hemoglobin 199
 - Fetal hypoglycemia 138
 - Fetal hypoxia 12, 128
 - Fetal loss 136
 - Fetal malformations 137
 - Fetal malposition 145
 - Fetal monitoring 138, 330
 - during labor 128
 - Fetal surgery, role of 164
 - Fetal tachycardia 138
 - Fetoscopic endoluminal tracheal occlusion 164
 - Fetus, effect on developing 136
 - Fiberoptic bronchoscope 182, 257, 308, 468
 - Fiberoptic intubation 180, 181, 581
 - Fibrinogen 147, 231
 - Fibrinolysis 14, 150
 - Fictitious hyponatremia 382
 - First aid 196
 - initial 204
 - First heart sound 54
 - Flagg's can 438
 - hazards of 438
 - Flail chest 196
 - Flattening test 409
 - Flatulence 149
 - Flexible laryngeal mask airway 539*f*
 - Flow vaporizer 434
 - Flow volume loops 307
 - Flowmeter 419
 - assembly 419
 - functioning of 419*f*
 - in anesthetic machine, arrangement of 420*f*
 - uses of 421*f*
 - with bobbins 419
 - Flow-over vaporizer 434
 - Flow-volume loop 96*f*
 - advantages of 401
 - Fluid and electrolyte
 - changes 106
 - derangement 332
 - Fluid balance 241
 - Fluid management 368
 - plan for 83
 - Fluid regimen, perioperative 73
 - Fluid restriction 83, 196
 - Fluid resuscitation 197, 325
 - complications of 198
 - formulas for 198*t*
 - Fluid therapy 323
 - manage 170
 - rate of 323
 - Fluorescence in situ hybridization 209
 - Foam-filled cuffs 464
 - Focal neurodeficit, development of 172
 - Folate deficiency 118
 - Folic acid
 - rich foods 119
 - supplements 119
 - Fondaparinux 242
 - Foramen of Magendie 151
 - Foramina of Luschka 151
 - Forced expiratory
 - flow 88
 - time 92
 - volume 77
 - Forced vital capacity 77, 88, 252, 402, 403
 - Foreign body 527
 - airway obstruction 483
 - Fossa lesions, posterior 172
 - Four-chamber pleural drain system 102
 - Fracture
 - closed reduction of 288
 - management of 288
 - neck femur 236
 - Frangible disk assembly 411
 - Frank-Starling curve 278
 - Free radical formation 168
 - Fresh frozen plasma 143, 147, 237, 296, 553
 - Fresh gas
 - compensation 428
 - decoupling 428
 - advantages of 428
 - disadvantages of 428
 - flow 421, 452, 453, 455*f*, 457*f*-460*f*
 - Fuji uniblocker 472, 472*f*
 - Full bladder 41
 - Full blood count 49, 211, 336
 - Full stomach patient 124
 - Fulminant liver failure 277
 - Functional brain reserve 217
 - Functional residual capacity 49, 124, 215, 252
 - Furosemide 10, 46, 156
 - Fusible plug 411
- G**
- Gabapentin 204
 - Gait difficulties 153
 - Galactosemia 245
 - Gallbladder surgery 187
 - Gamma aminobutyric acid 271
 - Ganglion-blocking agents 322
 - Gas
 - advantages of 280, 280*t*
 - and vapor 407
 - concentrations 433
 - cylinder 409
 - dilution techniques 399
 - disadvantages of 280, 280*t*
 - embolism 283
 - exchange 126, 216
 - flow 446
 - around bobbin 419
 - height of 435
 - laws 431
 - properties of 280
 - under diaphragm 530*f*
 - Gaseous induction 156
 - Gas-liquid interface 434
 - Gastrectomy, total 403
 - Gastric acid aspiration 136
 - Gastric
 - banding 259
 - catheter 167
 - emptying 156
 - port 271
 - Gastroesophageal reflux disease 130, 347
 - Gastrointestinal abnormalities 334
 - Gastrointestinal effects 282
 - Gastrointestinal system 94, 218, 303, 315
 - Gastrointestinal tract 194, 470
 - Gastroparesis 317
 - Gay-Lussac's law 431
 - General anesthesia 8, 65, 68, 131, 138, 148, 174, 189*f*, 222, 238, 262, 338, 347
 - administering 350
 - comparison of 148*t*
 - considerations for 261
 - induction of 163
 - technique for 138
 - Genetic disorders 279
 - Genetic factors 53
 - Genetic predisposition 105
 - Genitourinary complications 160
 - Gentamicin 12
 - Geriatric patient 214, 236
 - Gestation, duration of 108
 - Gestational diabetes mellitus 313, 328
 - classify 329
 - diagnostic criteria for 328
 - pathophysiology of 328
 - Gestational diabetes, subtypes of 329
 - Gestational hypertension 104
 - Glargine 318
 - Glasgow coma
 - scale 166*t*, 167, 195
 - score 165
 - Glaucoma, lens-induced 244
 - Glidescope 585*f*
 - types of 585, 585*t*
 - Glisson's capsule 104
 - Glomerular filtration rate 217
 - Glomerulonephritis, acute 42
 - Glottic opening
 - score, percentage of 176, 176*f*
 - size of 183
 - Glucagon-like peptide-1 analogs 318
 - Gluconeogenesis 264
 - Glucose 229
 - supplementation 321
 - synthesis 264
 - Glucose-insulin-potassium infusions 325
 - Glycemic control 171
 - Glycine 229
 - toxicity 230
 - signs of 230
 - Glycogenolysis 264, 319
 - Glycopyrrolate 220, 360
 - Glycosaminoglycans 152
 - Glycosylated hemoglobin 320, 321
 - Goal-directed fluid therapy 278
 - Goiter 402
 - large 183
 - Goldenhar syndrome 208
 - Goldman's vaporizer 436, 443, 443*t*
 - output of 443
 - Graham's law of diffusion 431
 - Graham's steel murmur 5
 - Gram's stain 87
 - Graves' disease 308, 312
 - Gravida's increased basal metabolic rate 124
 - Greene needle 551
 - Guanosine monophosphate 217
 - Guedel airway 490
 - Gum elastic bougie 114*t*
 - Gupta perioperative cardiac risk calculator 22
 - Gurd's criteria 239
 - Gynecological surgeries, robotic-assisted 286
 - Gynecomastia 273
- H**
- Haemophilus influenzae* 86
 - Hagen-Poiseuille equation 421
 - Hair follicles 191
 - Haloperidol 225
 - Halothane hepatitis, pathophysiology of 277
 - Hanger yoke assembly 413

- Hartmann's solution 212
- Head
 - injury 169, 170
 - trauma 42
- Headache 141, 199
 - severe 42
- Headbox oxygen 574f
- Hearing
 - loss 209
 - problems 152
- Heart 38, 214
 - block
 - complete 52, 513f
 - first-degree 512
 - disease 129
 - congenital 45, 52, 163
 - embryology of 53
 - failure
 - congestive 4, 55, 516
 - symptoms 61
 - normal 46f
 - rate 70
 - calculation of 505f, 506f
 - variability 316
 - sound, second 54
 - strain, right 75
 - valve, artificial 532
- Heartburn during pregnancy 124
- Heidbrink valve 451, 452f, 456
- Helium 410
 - dilution technique 399
- HELLP syndrome 107, 116, 118
- Hemabate 149
- Hematologic disorders 136
- Hematologic system 193
- Hematological abnormalities 294
- Hematological and immune system 219
- Hematological changes 141
- Hematoma 131
 - block 288
- Hemiblock
 - left anterior 511f
 - left posterior 511
- Hemifacial microsomia 208
- Hemodynamic changes during laparoscopy 281
- Hemodynamic collapse, causes of 283
- Hemodynamic instability 522
- Hemodynamic management 169
- Hemodynamic monitoring, close 12
- Hemoglobin 119, 307, 320, 401
 - estimation 120
- Hemolysis 231
- Hemolytic jaundice 266
- Hemoptysis, causes of 4
- Hemorrhage 84, 166, 239, 283
 - antepartum 143, 144
 - by class
 - signs of 167t
 - symptoms of 167t
 - causes of 143
 - obstetric 143
 - retrobulbar 247
 - subarachnoid 129
- Hemorrhagic shock 166
- Hemostatic system 93
- Hemothorax 196
- Henry's law 431
- Heparin, unfractionated 10, 13, 242
- Hepatic blood flow 282
 - reduced 218
 - regulation of 264
- Hepatic clearance 366
- Hepatic drug clearance 277
- Hepatic encephalopathy 271
 - grades of 272t
- Hepatic system 218
- Hepatitis
 - B 266, 267
 - virus 267
 - C 266, 267
 - virus 267
 - viruses 118
 - types of 267t
- Hepatocellular necrosis 279
- Hepatocyte 266
- Hepatorenal syndrome 269
- Hepatotoxicity 304
- Hereditary 118
- Hernia 276
- High airway pressure 579
- High intrinsic clearance drugs 277
- High minute ventilation 390
- High oxygen concentration 187
- High pressure alarm 390
- High-flow systems 575
- High-pressure cuff 464
- High-volume low-pressure cuff 464
- Hip
 - dislocation 162
 - fractures, types of 236
 - protectors 243
- His-Purkinje system 61
- Histamine 193, 204
- Histidine-tryptophan-ketoglutarate solution 340
- Holliday-Segar formula 212
- Hormonal synthesis 126
- Horner's syndrome 290
- Hospital cardiac arrest 481
- Human immunodeficiency virus 118, 267
 - transmission, risk of 268f
- Humphrey's ADE system 461
- Hunter-Hurler phenotype 3
- Hyaline membrane disease 53
- Hydralazine 114
 - hydrochloride 43
- Hydration and mucolytic therapy 79
- Hydrocephalus 151, 164
 - active 153
 - acute 163
 - chronic 151, 153
 - etiological classification of 151
 - presentation of 152
 - signs of 153, 162
 - symptoms of 153
- Hydrofluoric acid 204, 205
- Hydrogen
 - cyanide 200
 - peroxide 301
- Hydropneumothorax 529f
- Hydroxybutyrate 322
- Hyomental distance 174
- Hyperalgesia 204
 - primary 204
 - secondary 204
- Hyperbaric hyperoxia 376
- Hyperbaric oxygen, role of 200
- Hypercalcemia 524
- Hypercapnia 49, 73, 136, 284
 - effects of 281
- Hypercarbia 8, 41, 223, 278
- Hyperglycemia 314, 320, 326, 382
 - adverse effects of 320
 - severe 63
 - symptoms of 322
- Hyperglycemic emergencies 322
- Hyperkalemia 63, 146, 524, 525f
 - manage acute 338
 - progressive changes of 524
- Hypermagnesemia 115
- Hyperosmolar hyperglycemic state 322, 323
 - treatment of 323
- Hyperosmolar therapy 170, 173
- Hyperostosis cranii ex vacuo 155
- Hyperoxia 376
- Hypersensitivity 278
- Hypersomnolence 252
- Hypertension 35, 37, 40, 41, 73, 214, 215, 221, 222, 226, 251, 261
 - categorization of 35t
 - cause of 37
 - intraoperative 40
 - postoperative 41
 - chronic 105
 - control 38, 72
 - isolated systolic 40
 - malignant 40, 42, 129
 - pathophysiology of essential 38
 - postoperative 42
 - secondary 37
 - severe 41
 - uncontrolled 41, 43
- Hypertensive carotid sinus 58
- Hypertensive crisis 42
- Hypertensive disorders 104
- Hypertensive emergencies, treatment of 43t
- Hypertensive encephalopathy 42
- Hypertensive pulmonary vascular disease 55
- Hyperthermia, avoid 171
- Hyperthyroid, management in 304
- Hyperthyroidism 40, 525
 - causes of 303
 - diagnosis of 303t
 - pregnancy in 312
 - symptoms of 302
- Hypertonic saline acts 170
- Hypertrophic cardiomyopathies 214
- Hyperventilation 109, 138, 157
- Hypervolemia 8, 41, 368
- Hypo- and hyperthyroidism, signs of 302
- Hypocalcemia 146, 223, 311, 333, 524
- Hypocapnia, mild 157, 158, 169
- Hypodermis 192
- Hypoglycaemia, risk of 321
- Hypokalemia 223, 524
- Hypomagnesemia 223
- Hyponatremia 170
- Hypoplastic anemia 118
- Hypotension 73, 113, 138, 148, 222, 321
 - causes of intraoperative 239
 - higher incidence of 26
 - induced 295
- Hypothalamic-pituitary-adrenal 80
 - testing 342
- Hypothermia 63, 73, 74, 146, 196, 201, 217, 224, 232, 279, 525f
 - mild 224
 - prevention of 217
 - severe 482
- Hypothyroid, anesthesia in 306
- Hypothyroidism 63, 72, 306
 - causes of 305
 - changes in 524
 - diagnosis of 303t
 - implications of 306
 - signs of 302
 - symptoms of 302
- Hypotonic fluids, administration of 223
- Hypoventilation 138
- Hypovolemia 7, 74, 212
 - signs of 197
 - uncorrected 110
- Hypovolemic shock 197
- Hypoxemia 49, 103, 278, 566
 - signs of 197

- Hypoxemic episodes 73
 Hypoxia 8, 49, 97, 136, 223, 566
 causes of 566
 chronic 46
 low-flow 199
 recurrent episodes of 91
 severe 63
 Hypoxic mixture 418
 delivery of 418, 419
 Hypoxic neuronal edema 327
 Hypoxic pulmonary vasoconstrictor 216
 Hypoxic spells 46
 Hysteresis 60
- I**
- Icterus 265
 Ideal nerve stimulator 560
 Idiopathic autonomic neuropathy 316
 Idiosyncratic drug reactions 278
 I-gel supraglottic airway 545, 545f
 Ileus 300
 Immune
 function 334
 system responses 193
 Immunoglobulin 267
 Impedance threshold device 484
 Impending eclampsia, signs of 113
 Implanted cardioverter defibrillator 68
 Inadvertent brainstem anesthesia 247
 Incentive spirometry 79
 Incompetent cervix 136
 Incontinentia pigmenti 53
 Indirect acting valves 411t
 Indomethacin 55, 138
 Induction agents 322
 Indwelling pleural catheter 102
 Infarction 63, 73
 Infection 155, 231
 prophylaxis 172
 Infectious causes 136
 Infectious ventriculitis 152
 Infective endocarditis 49, 50, 52
 prophylaxis 50*
 role of 6
 Inferior wall myocardial infarction 521f
 Inflammatory mediators 204, 368
 Inflating system 463
 Influenza vaccine 88
 Infralavicular brachial plexus block 289
 Intrainguinal arterial bypass 73
 Infraorbital nerve block 213f
 Infusions, target-controlled 366, 367
 Inhalation agent 49, 127, 220, 257, 277, 335
 concentrations of 170
 effect of 134
 Inhalational induction 156
 Inhalational injuries
 diagnose 195
 diagnosis of 194
 pathophysiology of 194
 Inhalers 436
 Inherited metabolic liver diseases 267
 In-hospital cardiac arrest 481
 Initiate ammonia-lowering therapy 274
 Injection type vaporizers 448
 Injuries, type of 205
 Insomnia 152
 Inspiration pressure regulator 577
 Inspiratory cycling 385
 Inspiratory phase 387
 Inspired oxygen, fraction of 390
 Insufflation 280
- Insulin 318
 correctional 326
 deficiency 322
 infusion, intraoperative short-acting 321
 preparations 318
 rapid-acting 329
 resistance syndrome 314
 sensitivity factor 326
 short-acting 319
 supplementation 318
 therapy 323
 future of 319
 types of 318
 Insulin-dependent diabetes mellitus 161, 383
 Insulin-mediated peripheral glucose 238
 In-system vaporizers 436
 Intensive care monitoring 142
 Intensive care unit 154, 480, 526
 intubation protocol 190
 Intercostal drain 99
 Intercostal nerve blocks 84
 Intermediate-acting preparations 318
 Intermittent blood pressure 138
 Intermittent flow anesthesia machines 428
 Intermittent mandatory ventilation 387
 Intermittent pneumatic calf compression devices 259
 Intermittent positive pressure ventilation 80, 169, 258, 309, 463, 492
 Interscalene block 558t
 Interscalene brachial plexus block 290
 Interventional cardiac surgery 56
 Intestinal obstruction 324
 Intestine 69
 Intra-abdominal
 pressure 280, 281
 surgery 370
 Intracerebral bleeding 140
 Intracranial bleeding 42
 Intracranial hypertension, management of 171
 Intracranial pressure 40, 151, 166
 increased 390
 raised 180
 Intracranial tumor 152
 Intracuff pressure 464
 Intrahepatic cholestasis 279
 Intraneural injection 557
 Intraocular pressure 180
 Intraocular tension 244
 Intraoral injection 212
 Intrapleural pressures 52
 Intrapulmonary right-to-left shunt 98
 Intraspinous pressure 299
 Intrathecal opioids 84, 356
 Intrathoracic obstruction 403f
 Intratracheal extension 301
 Intratracheal placement 466
 Intrauterine diagnosis 161
 Intrauterine growth retardation 107
 Intravenous
 antihypertensive agents 44
 calcium 150
 crystalloid fluids 165
 fluids 241, 321
 induction 156
 lipid emulsion 142
 narcotic 166
 regional anesthesia 289
 Intraventricular hemorrhage 152, 153
 Intrinsic heart rate 62
 Intubating laryngeal
 airway 540
 mask airway 178, 181, 540
 Invasive arterial
 blood pressure monitoring 239
 pressure 278
- Iodides 304
 Iodine deficiency, severe 305
 Ionization, degree of 126
 Iron
 deficiency anemia 118, 120
 supplements 119
 therapy, parenteral route of 121
 Iron-dextran 121
 Iron-rich foods 119
 Iron-sorbitol-citric acid complex 121
 Irritable uterine activity 138
 Ischemia 117
 cold 340
 perioperative 19
 warm 340
 Ischemic heart disease 17, 28, 36, 75, 214, 226, 321, 333
 development of 17
 diagnosis of 20
 evaluation of 24f
 Ischemic optic neuropathy 286, 299t
 Ischemic pain mechanism 354
 Ischial spine 353
 Isoflurane 335, 425
 Isotope scanning 72
- J**
- Jackson-Rees circuit 460, 460f
 Jaundice 265
 causes of postoperative 279
 cholestatic 266
 haemolytic 266, 266t
 hepatocellular 266, 266t
 obstructive 265, 266, 266t
 Joffroy's sign 302
 Joint 218
 mobility 217
 national committee 35
 Judicious fluid therapy 7
- K**
- Kepler intubation system 591, 591f
 Ketamine 163, 184, 203, 357
 Ketone bodies 324
 Ketotifen 249
 Kidney 38, 69, 106
 cocktail 340
 disease 344
 chronic 35, 332
 enlargement 162
 role of 376
 Kinemyography 561
 King Vision videolaryngoscope 590, 590f
 Kissing tonsils 348
 Klippel-Feil syndrome 209
 Knee-chest position 47
 Kyphoscoliosis 293, 294, 397
 correction surgery 294
 surgery 294
 Kyphosis 293
- L**
- Labetalol 108, 114
 hydrochloride 43
 Labor
 analgesia, techniques of 109, 110
 and delivery, diabetes for 330
 pain, adverse effects of 109
 process 128
 prolonged 112

- Lack's circuit 456
- Lack's coaxial
 - circuit, testing 456
 - system 456, 456f
- Lactic acidosis 317
- Laerdal resuscitator 578
- Laerdal valve 502f
- Lancinating pain 112
- Laparoscopic cholecystectomy 280, 283, 284
- Laparoscopic surgery 280, 285, 392
 - advantages of 280
- Laparoscopy 280, 283
 - advantages 284
 - contraindications for 285
 - pneumoperitoneum for 282
 - respiratory changes during 281
 - safe during pregnancy 284
 - technique of 280
 - ventilatory changes during 281
- Laparotomy, evacuation of 170
- Largactil 114
- Large cylinders, connections for 412
- Large-bore cannulas 143
- Laryngeal edema 311, 465
- Laryngeal manipulation, optimal external 130, 185
- Laryngeal mask airway 95, 114, 130, 178, 181, 182, 201, 248, 309, 348, 351, 478, 534, 534f, 536f, 539f, 540, 547
 - adverse effects of 536
 - causes of 536
 - classic excel 539
 - complications of 536
 - contraindications for 536
 - flexible 539
 - gastro airway 547f
 - indications for 536
 - insertion, technique of 535, 535f
 - protector 547f
 - seal pressure for 536
 - single use 537f
 - supreme 538, 538f
 - unique 539
- Laryngeal nerve
 - block, superior 180
 - recurrent 312
- Laryngeal tube 478, 540, 541, 542f
 - suction 541, 542f
- Laryngoscope 494, 495
 - blade 130
 - conventional 583
 - indirect 581
 - malfunction 497
 - parts of 494
- Laryngoscopy
 - complications of 497
 - intubation 132
 - technique 496
- Laryngospasm 311
- Larynx, nerve supply of 311
- Latanoprost 249
- Lateral gaze palsy 153
- Leaflet 5
 - calcification 8
 - mobility 8
 - thickness 8
- Lean body mass 260
- Leboyer technique 109
- Lee's revised cardiac risk index 71
- Left atrial
 - contraction 3
 - to aortic root 54
- Left atrium 59f
- Left bundle branch block 58, 511, 511f, 522
- Left double-lumen tube 471f
- Left lung transplantation 469
- Left ventricular
 - dysfunction 39
 - ejection fraction 61
 - hypertrophy 36, 251, 509, 519f
 - causes for 519
 - systolic function 54
- Leiomyomas 136
- Lesion
 - arising 183
 - management of 162
 - pressure of 162
- Lethargy 199
- Leukocytosis 236
- Leukotrienes 204
- Levobupivacaine 111, 352
- Lewis-Leigh valve 502
- Life-threatening
 - infection 219
 - obstetric emergency 140
- Ligamentum flavum, calcification of 217
- Light discriminatory touch 204
- Light headedness 113
- Lightning electrical burn 205
- Lignocaine 115, 180, 288, 289
 - jelly 180
- Limb compartment syndromes 299
- Lindeque's criteria 239
- Linder nasopharyngeal airway 493
- Lipid
 - emulsion, administration of 113
 - peroxidation 168
 - profile 71
 - solubility 126
- Lipid-lowering drugs 27
- Lipophilic drugs, adsorption of 260
- Lipoprotein, low-density 70
- Lippman-Cobb method 293, 293f
- Liquefied gases 410
- Liquid chemical disinfection 475
- Liquid-crystal display 448, 586f
- Lithium
 - dilution cardiac output 239
 - hydroxide 425
- Lithotomy position
 - complications of 227
 - variations of 227
- Liver 107, 227
 - biosynthetic function of 265
 - cell failure, signs of 273
 - cirrhosis, chronic 273
 - disease 276
 - chronic 266, 273
 - staging of 273
 - enzymes 107, 266
 - elevated 116
 - function test 87, 265
 - functions of 264
 - laceration 170
 - tissue 218
- Lobar collapse 531
 - treatment of postoperative 531
- Lobectomy 78, 395
- Local anesthesia 127, 204, 221, 350
 - clinical 203
- Local anesthetic
 - injection of 558
 - placement of 180
 - toxicity, signs of 292
- Local blocks, advantages of 245
- Local toxicity 113
- Longer-acting insulin preparations 318
- Lorazepam 220, 233, 292
- Lordosis 293
- Lose oxygen pipeline pressure 427
- Low concentrations, combination of 223
- Low dietary calcium intake 106
- Low filling port 438
- Low minute ventilation 390
- Low molecular weight heparin 13, 106, 242
- Low pressure alarm 390
- Lower limb 558
 - amputees 594
 - motor supply areas of 558t
- Lower segment cesarean section 131, 143
 - classification of 131t
 - emergency 128
- Low-flow anesthesia
 - advantages of 426
 - disadvantages of 426
- Low-flow oxygen delivery system 568
- Low-molecular-weight heparin 10, 172
- Lown-Ganong-Levine syndrome 507, 509f
- Lumbar sympathetic block, role of 73
- Lumboperitoneal shunt 154
- Lund and Browder chart 191
- Lung 69
 - agenesis 529
 - capacity, total 402
 - collapse 529
 - dependent 83
 - function 76
 - postoperative 76, 77
 - isolation 81
 - advantages 81, 81t
 - devices 462
 - disadvantages 81, 81t
 - manipulation, intraoperative 83
 - maturity 108
 - mechanical function 76
 - methods of isolation of 474
 - movement 187
 - nondependent 83
 - parenchyma
 - function 76
 - loss of 75
 - pathology 398
 - resection 75, 79f
 - surgery 75
 - scan 375
 - separation, indications for 80
 - subsegments of 76f
 - volumes and capacities 215
- Luteal-phase deficiency 136
- Lutembacher's syndrome 6
- Lymph, formation of 264
- Lysosomal enzymes 152
- Lytic cocktail 114

M

- Macewen's sign 152, 158
- Macintosh blade 495f
- Macrocytic anemia 118
- Macrosomia 329
- Magill's system 456
- Magnesium 333
 - sulfate 115, 478
- Magnet
 - application, effect of 62
 - use of 66
- Malaise 199
- Mallampati classification 174
- Malleable intubation stylet 470
- Mandibular advancement devices 261
- Mandibular nerve 562
- Mannitol 157, 229

- Manual resuscitator 566, 575, 576, 576f, 578
use of 575, 579
- Manual uterine displacement 141
- Mapleson A
breathing system 455
system 455
- Mapleson B and C system 457
- Mapleson breathing systems 454f
- Mapleson circuit 455f
controlled ventilation in 456f
- Mapleson D system 458
- Mapleson E system 457
- Mapleson's breathing systems
advantages of 455
disadvantages of 455
- Mapleson's classification 454
- Mapleson's systems 456
ranking of 455
- Marfan's syndrome 152
- Marshall scale 170
- Mask
seizure activity 171
ventilation, difficult 176
- Massive blood
loss 296
transfusion, complications of 296, 296f
- Massive obstetric hemorrhage 144, 146
treatment options for 146
- Massive pulmonary
edema 129
embolism 521
- Massive transfusion 296
management of 296
protocol 296
- Master and slave mechanism 417
- Maternal cardiovascular risk, classification of 15t
- Maternal complications 108, 146
- Maternal concerns 136
- Maternal fetal concentration gradient 127
- Maternal hematocrit 11
- Maternal hemoglobin 109
- Maternal hypertension 144
- Maternal hyperventilation 109
- Maternal hypothermia 161
- Maternal refusal 110
- Maternal safety 136
- Maximal voluntary ventilation 88
- Maximum expiratory flow 402, 403
- Maximum laryngeal height 92
- Maximum voluntary ventilation 398
- McCoy laryngoscope 495
- McGrath laryngoscope 585
- McGrath videolaryngoscope 586f
- Mean arterial pressure 38, 299
- Mean corpuscular volume 118
- Measured-flow vaporizer 434f
- Mechanical prosthetic heart valve 14
- Mechanical thumbs 385
- Mechanical valve 9
prostheses 13
types of 9
- Mechanical ventilation 385
complications of 390
- Mechanomyography 561
- Meconium, utero passage of 129
- Meconium-stained amniotic fluid, implications
of 129
- Mediastinal mass 531, 531f
anterior 184
excision 286
- Medical devices directive 462
- Medium dependency system 573
- Medulloblastoma 151
- Megaloblastic anemia 120
- Meglitinides 317, 319
- Meissner's corpuscles 204
- Memory 216
- Mendelian's disorders 136
- Mendelson's syndrome 129
prevention of 129
- Meningeal signs 158
- Meningioma 151
- Meningitis 151
- Meningocele 161
- Meningomyelocele 152, 160, 161
management of child with 162
- Mental decline 153
- Mental foramina 212
- Meperidine 336
- Mercury-zinc batteries 59
- Mesenteric artery syndrome, superior 300
- Metabolic acidosis 49, 379-381, 384
causes of 380
simple 382
- Metabolic alkalosis 379
causes of 381
- Metabolic disturbances 311
- Metabolic equivalents 71
- Metabolic functions 264
- Metabolic responses 193
- Metabolic syndrome 314
- Metabolic-idiopathic 278
- Metabolism 218, 265, 277, 302
- Metformin 319
- Methyl methacrylate 240
- Methyldopa 107
- Methysergide therapy 3
- Metoclopramide decreases gastric volume,
administration of 129
- Metoprolol 8, 40
- Microalbuminuria, urine routine for 320
- Microangiopathic hemolysis 104
- Microlaryngeal tracheal surgery tubes 468
- Microlaryngeal tube 468f
- Microspherocytes 120
- Midazolam 201, 220, 258, 260, 321
metabolites of 276
- Mid-diastolic murmur, differential diagnosis for 5
- Miller blade 495
- Miller's supraglottic classification 540
- Minimal alveolar concentration 126
- Minimally invasive direct cardiac massage 485
- Minimum alveolar concentration 134, 204
- Minimum mandatory oxygen flow 418
- Minute ventilation 388, 453, 595
change in 281
- Minute volume dividers 386
- Misoprostol 141, 149
side effects 149
- Mitral annular calcification 3
- Mitral commissurotomy 11
closed 11
- Mitral regurgitation, moderate-to-severe 6
- Mitral stenosis 3, 5, 9, 532, 532f
causes of 3
grades of 4t
pathophysiology of 3
signs of 4
symptoms of 4, 9
- Mitral valve
anatomy of 5
prolapse 210
repair, indications for 9
replacement, indications for 9
- Mivacurium 260
- M-mode
echocardiography 54
technique 6
- Monomorphic ventricular tachycardia 518f
- Monro-Kellie doctrine 169f
- Morbid obesity 110, 250
- Morbidly obese 111
- Morphine 112, 127, 261, 277, 336, 360
- Mortality, risk factors and 21t
- Mother's well-being 261
- Moustache sign 5
- Mucopolysaccharidoses 152
- Multifactorial disorders 136
- Multifunction chest drainage system 102, 102f
- Multimodal analgesia 355
- Multiorgan failure 199
- Multipara 144
- Multiple parameter tests 175
- Multiple pregnancy 144
- Murmur 5
benign 210
- Murphy's eye 463, 468, 587
- Muscle
block 285
contraction 592
relaxants 127, 203, 220, 276
relaxation, reversal of 96
- Muscular contraction 561
- Muscular fatigue 562
- Musculoskeletal defects 161
- Musculoskeletal disorders 163
- Musculoskeletal system 126, 218
- Mycobacterium*
avium complex 86
tuberculosis 86
- Mycophenolate mofetil 341
- Mydriatics 249
- Myocardial burns 65
- Myocardial contractility 18
- Myocardial infarction 28, 69, 117, 221, 519
acute 42, 140
after 58
management of perioperative 32
perioperative 18
- Myocardial injury 32
- Myocardial ischemia 42, 63, 71, 73, 74
clinical manifestations of 18
pathophysiology of 18
risk of 73
- Myocardial oxygen
delivery 18
demand and delivery 18
supply and demand, causes of imbalance in
30t
supply-demand balance 29
- Myocardium 63, 69, 214
- Myoclonic movements 65
- Myoglobin 199
- Myomectomy, previous 144
- Myopic eye, severely 247
- Myxedema coma 305
- N**
- N-acetylcysteine 343
- Naloxone 115
- Narcotic 257
analgesics 197
- Nasal airway 492
measurement of 492f
- Nasal cannula 568, 569f, 574f
advantages of 569
disadvantages of 569
high-flow 181
- Nasal catheter 574f
- Nasal high flow oxygenation 575
- Nasal intermittent positive pressure ventilation
259

- Nasal mucosa 194
- Nasal oxygen 187
 - technique of 187, 187f
- Nasal passage 179
- Nasal prongs 203
- Nasal regurgitation, history of 207
- Nasogastric
 - lavage 274
 - tube 129
- Nasopharyngeal airway 492
 - advantages of 492
 - description of 492
 - types of 493
- Nasopharyngeal catheter 574f
- Nasopharyngoscopy 213
- Nasopharynx 213
- Nausea 149, 199
 - postoperative 283, 356
- Near-infrared spectroscopy 55
- Nebulizer 570
 - face mask
 - advantages of 570
 - disadvantages of 570
- Neck
 - contracture, release of 203
 - mass, anterior 308
- Needle cricothyrotomy 186
- Needle insertion, techniques of 550
- Negative and positive electrodes, placement of 562f
- Neonatal complications
 - long-term 107
 - short-term 107
- Neonatal hypoglycaemia, risk of 329
- Neonatal resuscitation, protocol for 134
- Neoplasm invading trachea 402
- Neostigmine 260
 - dose of 220
- Nephropathy, contrast-induced 315
- Nephrotoxic agents 343
- Nerve locator 554, 555
 - advantages of 554
 - limitations of 554
 - use of 291
- Nervous system 107
- Neural defects, primary 161
- Neural tube defects 160, 160f, 161
- Neurally adjusted ventilatory assist 393
- Neuraxial block 220
 - procedures 217
- Neuraxial system 217
- Neuraxial technique 132
- Neurocardiac syndromes 58
- Neuroendocrine effects 282
- Neurologic complications 205
- Neurologic effects 282
- Neurologic status, determine 165
- Neurologic worsening, signs of 171
- Neurological abnormalities 334
- Neurological assessment 162
- Neurological changes 141
- Neurological dysfunction, signs of 156
- Neurological examination 162
- Neurological status 197
- Neuromuscular block
 - degree of 560, 552
 - maintenance of 115
 - reversal of 338
- Neuromuscular blockers 335
 - residual effect of 98
- Neuromuscular blocking agent 156, 310
- Neuromuscular disease 381
- Neuromuscular function 217
- Neuromuscular monitoring 556, 559, 561, 564, 565
- Neuromuscular problems 398
- Neuromuscular recovery, signs of postoperative 564
- Neuronal shrinkage 216
- Neuropathic pain 204
- Neurosurgical procedures, common 154
- Neurulation
 - primary 160
 - secondary 160
- Neutralizing agents 205
- Newborn, general assessment of 162
- Nicardipine hydrochloride 43
- Nifedipine 107, 116
- Nine panel plot 595f
- Nitrates 33
- Nitric oxide 105, 193
 - inhaled 142
- Nitrogen
 - dioxide 195
 - washout technique 399
- Nitroglycerin 40, 43, 360
- Nitrous oxide 49, 137, 138, 282, 410, 413, 442, 464
 - Adams regulator for 415
 - critical temperature of 407
 - cylinder 408
 - filling ratio for 410
 - use of 157
- Nocturnal desaturation 91
- Noncardiac surgery 19, 26, 27, 32, 50
 - urgent 26f
- Noncardiogenic pulmonary edema 141
- Nonleft surgery 213
- Noncystic fibrosis bronchiectasis 86
- Nonexercise stress 71
- Non-life-threatening orthopedic surgery 170
- Nonlinear pharmacokinetics 365
- Nonliquefied gases 409
- Nonobstetric causes 140
- Nonobstetric surgery 136
- Nonopioid analgesics 335
- Nonparticulate antacid 129
- Nonpharmacological techniques 109
- Nonpharmacological therapy 36
- Nonrebreathing
 - circuit 453t
 - masks 571
- Nonrebreathing valve 489, 498, 499, 577, 579
 - comparison chart for 502t
- Nonreturn valve 441
- Non-Seldinger technique 186
- Nonshockable rhythm 477
- Nonsteroidal anti-inflammatory drug 97, 100, 116, 134, 139, 158, 202, 218, 223, 227, 242, 258, 279, 284, 335, 347, 355, 553
 - role of 355
 - transfer of 134
- Nontransplant surgery 341
- Noradrenaline 12, 107, 169
- Norepinephrine 216
- Normal pressure hydrocephalus 153
- Normobaric hyperoxia 376
- Normocarbica 138
- Normochromic normocytic anemia 120
- Normotensive pregnancy 106
- Normothermia 157, 222, 295
- Normovolemic hemodilution, acute 295
- Normoxia 222, 223
- Norpethidine 110
- Nutrient supply 126
- Nutrition 241
- O**
- O'Brien method 247
- Obesity 215, 250
- cardiomyopathy 251
 - categories of 250t
 - hypoventilation syndrome 251, 252
- Obstetric causes 140
- Obstetric haemorrhage
 - causes of 144
 - management of 149
- Obstetric interventions 132
- Obstructive lung disease 398, 401
- Obstructive pulmonary disease 92t
 - causes of chronic 90
 - chronic 70, 75, 90, 91, 91t, 97, 98, 284, 389, 399, 595
 - diagnose chronic 90
- Obstructive pulmonary function tests, causes of 398
- Obstructive sleep apnea 179, 188, 215, 252, 347
- Obturator nerve 228
- Ocular drugs, systemic effects of 248t
- Ocular effects 282
- Oculocardiac reflex 248
- Oculomotor nerve 153
- Ofloxacin 249
- Ogston's inhaler 438
- Oligohydramnios 139
- Oliguria 368
- Olopatadine 249
- One-compartment model 362f
- One-lung anesthesia, indications of 474
- One-lung ventilation 80, 83
 - management of 83
- Opaque hemithorax 529f
- Open commissurotomy 11
- Open heart surgery 328
- Open-chest cardiopulmonary resuscitation 484
- Operating room preparation 49
- Ophthalmic drugs, systemic effects of 248
- Opiate anesthetic techniques, high-dose 322
- Opioid 95, 109, 127, 204, 220
 - analgesics 336
- Opioid-dependent patients, manage 357
- Optic nerve 153
- Optimal laryngoscope blade 185
- Optimize fetal outcome 115
- Oral airway 177
- Oral anticoagulants 13
- Oral anticoagulants, novel 237
- Oral bioavailability 361f
- Oral iron 121
- Oral nifedipine 114
- Orbital fat 245
- Organ ischemia, risk factors for 294
- Organ preservation, used for 340
- Orogastric tube 129
- Oropharyngeal airway 248, 489, 489f, 490, 492
 - types of 490
- Oropharynx 185
 - for laryngoscopy 174
- Orthostatic hypotension 317
- Ortner's syndrome 5
- Oscillatory ventilation, high-frequency 393
- Osmolar gap 382
- Osmotic diuresis 171
- Osteoarthritis 221
- Osteoporosis 218, 221
 - treatment 242
- Ovassapian airway 491f
- Ovassapian fiberoptic intubating airway 491
- Overhydration 229
 - syndrome 230
- Oxford inflating bellows 441
- Oxford miniature vaporizer 440
- Oxford tube 467, 467f
- Oxidative deamination 230

- Oxiport macintosh 496
 Oxygen 33, 241, 408, 410, 427, 442, 592
 administration 574*f*
 consumption 124
 critical temperature of 407
 deficiency 195
 enrichment 573
 failure protection device 417
 flush valve 416
 hazards of 416
 free radicals 195
 hoods 574
 less 452
 nitrous oxide mixture 321
 pressure failure 417
 safety devices 417
 radicals 193
 source 187
 supplementation 325
 supply pressure failure 417
 tents 574
 therapy 98, 237, 566
 device 566-568, 575
 indications for 566
 toxicity 391
 prevents 575
 transport and utilization 592*f*
 Oxygenated blood 52, 57
 Oxygenation 375, 382
 Oxyhemoglobin dissociation 199
 Oxytocin 149
 side effects 149
- P**
- Paced heart 62
 Pacemaker 64, 66, 532, 532*f*
 artificial 58
 biventricular 61
 evaluation of 61
 failure 66
 function of 62
 generic codes of 59*t*
 insertion of 68
 leadless 68
 modern-day 59
 nomenclature 234
 permanent 58, 234
 programmable 61
 switch 62
 syndrome 61
 types of 58, 59*f*, 62
 Pacing malfunction 68
 Pacing threshold 60
 factors affecting 63*t*
 Pacinian corpuscles 204
 Pain 41
 acute postoperative 354
 causes of postoperative 354
 chronic postsurgical 354
 control postoperative 351
 fast 204
 incisional 283
 manage
 chronic 204
 postoperative 158
 pathophysiology of postoperative 354
 predicting postoperative 357
 relief 33
 type of 204
 Pain management 197, 204
 advances in 357
 barriers to 357
 perioperative 31
 Painful labor, adverse effects of 109
 Palatal cleft, type of 208
 Palate surgery, anesthesia for 207
 Palliative shunt 50
 Palliative surgery 47
 Pallor 148
 Palm print test 315
 Palmar erythema 273
 Palmar surface 191
 Palpitations 113
 Pancreatitis 300
 Pancuronium 219, 276
 Papillary muscles 5
 Parabulbar block 247
 Paracetamol 258, 261
 role of 355
 Parasternal heave, right 5
 Parasympathetic control, tests for 316
 Parathormone 333
 Parathyroid hyperplasia 301
 Paravertebral blocks 84
 Parenchymal disease 4
 Parenchymal function 78
 Parenchymal lung diseases 401
 Parenteral hydralazine 107
 Parenteral iron 121
 therapy, complications of 121
 Parenteral labetalol 107
 Parenteral opioids 84
 Parkinson's disease 153, 216
 Parkinsonism 221
 Parkland formula 197
 Paroxysmal supraventricular tachycardia 515, 516*f*
 Partial rebreathing mask 571
 Patellar reflex 114
 Patent ductus arteriosus 52, 52*f*, 210, 286
 Patent upper airway 187
 Patil endoscopic airway 491
 Patil-Syracuse oral airway 491, 491*f*
 Peak brain edema 172
 Peak expiratory flow 402, 403
 rate measurement 93
 Peak inspiratory flow rate 387
 Pemberton's sign 307
 Penicillin allergy 12
 Penile block 351
 Penlon bellows unit 441
 Penlon circuit 459, 459*f*
 Penlon coaxial circuit 459
 Pentax airway scope 588, 588*f*
 Percutaneous coronary intervention 25, 28, 237, 522
 Percutaneous mitral commissurotomy 8, 11
 Perforative peritonitis 327
 Perfusion index 55
 Peribulbar block 246
 Peribulbar needle position 246*f*
 Pericarditis 522
 Pericone 246
 Perimortem cesarean delivery 117
 decision for 117
 Periodic breathing during sleep 215
 Periorbital skin 246
 Peripheral arterial disease 70, 314
 asymptomatic 69
 Peripheral effects 113
 Peripheral nerve 554
 blocks 73, 221
 contraindications to 558
 technique of 557
 ultrasound guidance for 291
 injuries 228, 299
 stimulation, principles of 559
 stimulator 554, 559
 applications of 565
 Peripheral nervous system 217, 227
 Peripheral smear, study of 120
 Peripheral vascular disease 69, 70, 289
 anesthesia in 73
 medical treatment of 72
 Peripheral vasculature 38
 Periprocure analgesia 100
 Peritonsillar abscess 350
 Permanent pacemaker, indications of 58
 Permissive hyperglycemia 321
 Peroneal nerve, common 228, 562
 Peroxidases 199
 Pethick test 459
 Pethidine 114, 127, 219, 277, 336
 Phagocytic system 264
 Phagocytosis 327
 Pharmacokinetic pathway 360
 Pharmacologic agents 219
 Pharmacological compartments 361
 Pharmacological stress testing 21
 Pharmacological techniques 109
 Pharmacotherapy 35
 Pharyngeal sealers 541
 Phenergan 114
 Phenolphthalein 424
 Phentolamine 43
 Phenylephrine 12, 305
 Phenytoin 172, 365
 Pheochromocytoma 36, 40, 42
 Phonomyography 561
 Phosphate 324, 333
 Physiological abnormalities, management of 56
 Pierre Robin sequence 208
 and syndrome 209
 pathogenesis of 210
 Pierre Robin syndrome 210, 210*f*, 245
 Piezoelectric neuromuscular monitors 561
 Pilocarpine 249
 Pin index safety system 411, 412
 Pipeline pressure gauge 416
 Pipeline system, safety features in 415
 Placenta 107
 accreta 147
 grades of 147*f*
 accrete, management of 148
 delivery of 148
 increta 148
 normal 147*f*
 percreta 148
 previa 143, 144
 types of 144*f*
 removal of 115
 retained 146, 148
 Placental abruption 143, 144
 Placental blood sampling 164
 Placental peptides 106
 Placental separation, signs of 148
 Placental transport mechanisms 126
 Plasma albumin concentrations 203
 Plasma expanders 241
 Plasma magnesium 114
 levels 114*t*
 Plasma protein
 binding 260
 fraction 231
 Plaster of Paris 288
 Plastic disposable tubing 452
 Platelet 147
 count 70, 116
 dysfunction 116
 low 116
 Plenum vaporizers 436

- Plethora 252
 - Plethysmography 55
 - Pleural collections 99
 - Pleural effusion 99, 529f
 - Pleural fluid 529
 - Pleural pressure 400
 - Pneumatic compression devices 138
 - Pneumatic devices 418
 - Pneumococcal vaccine 88
 - Pneumectomy 75, 76, 78, 83, 84, 395, 529
 - left 78, 469
 - space 84
 - Pneumonia 48, 89, 368, 479
 - recurrent 53
 - Pneumonitis 479
 - Pneumoperitoneum 284
 - effects of 282
 - Pneumothorax 99, 103, 196, 283, 391, 528f
 - Polio blade 496
 - Polycythemia 52, 75, 402
 - predisposes 46
 - Polymorphonutrophil 327
 - Polysomnography 252
 - Polyurethane 200
 - cuffs 465
 - Polyvinyl chloride 181, 309, 462, 541
 - double-lumen tube 469
 - Popliteal nerve block 559t
 - Porcine 9
 - Porencephaly 152
 - Portal hypertension 268
 - causes of 269
 - complications of 269
 - management of 271
 - Portal sinus 52
 - Portopulmonary hypertension 270
 - Portosystemic anastomoses 269t
 - Portosystemic shunt procedures 271
 - Positive end-expiratory pressure 49, 130, 157, 188, 223, 389, 422
 - contraindications to 390
 - Positive intrathoracic pressure 390
 - Positive predictive value 175, 176, 179
 - Positive pressure ventilator 386
 - Positive-end expiratory pressure, adverse effects of 389
 - Postamniocentesis 144
 - Postanesthesia respiratory distress, causes of 311
 - Postdural puncture headache 112
 - Posterior wall myocardial infarction 521f
 - Postexposure prophylaxis 267, 268
 - Postoperative pain 356
 - management 50, 139, 355
 - Postpalatal cleft 208
 - Postpartum hemorrhage 108, 144, 145
 - primary 144
 - secondary 144
 - Postpneumectomy syndrome 84
 - Postrenal transplant 341, 342
 - Postsinusoidal intrahepatic causes 269
 - Postsinusoidal posthepatic causes 269
 - Postspinal bradycardia, treatment of 486
 - Post-tetanic
 - count 562
 - facilitation 564
 - Post-thoracotomy analgesia 84
 - Post-tonsillectomy pain 348
 - Postural drainage 88f, 171
 - Potassium 332
 - therapy 324
 - Potential toxicity 203
 - Poynting effect 413
 - Prayer sign 315
 - Predictable drug reactions 278
 - Pre-eclampsia 42, 104, 104f, 105-107, 115
 - development of 105
 - management of 108
 - severe 108
 - uncomplicated 108
 - medical management of 107
 - mild 104f
 - pathogenesis of 105
 - risk factors for 106
 - severe 104
 - severity of 104, 108
 - superimposed 105
 - Pre-eclamptic toxemia 104
 - Pregnancy
 - early 143
 - last half of 130
 - physiological anemia of 118
 - Prehepatic causes 269
 - Premature labor 136
 - risk of 139
 - Preoperative physiotherapy, role of 88
 - Preoxygenate 56, 183
 - Preserve spontaneous breathing 182
 - Presinusoidal intrahepatic causes 269
 - Pressor responses, increased 105
 - Pressure augmentation 393
 - Pressure gradient stockings 237
 - Pressure regulator 414, 415f
 - principle of 415
 - safety features on 415
 - Pressure sores, prevention of 237
 - Pressurizing effect, mechanism of 437
 - Presystolic accentuation of murmur, causes 5
 - Prilocaine 289
 - Primary care engagement 268
 - Procainamide 11
 - Procoagulants 265
 - Progressive neurological disease 160
 - Prokinetic agent 129, 137
 - Prominent U waves 507
 - Prophylactic antibiotic 156, 197
 - role of 130
 - therapy 12
 - Prophylactic antimicrobial therapy 12
 - Prophylactic epidural blood patch 112
 - Prophylactic treatment 112, 172
 - Propofol 79, 127, 220, 257, 258, 260, 335
 - Propranolol 360
 - Propylthiouracil 304
 - Proseal laryngeal mask airway 538, 538f
 - Prostacyclin 53
 - Prostaglandin 57, 105, 193, 204
 - analogs 249
 - primarily 53
 - Prostate 226
 - gland, anatomy of 226
 - syndrome, transurethral resection of 230, 232, 233
 - transurethral resection of 66, 226
 - Prostatic hyperplasia, benign 226
 - Prostatic resection, newer techniques of 234
 - Prosthetic aortic valve 12
 - Prosthetic thrombosis 14
 - Prosthetic valve 12, 532f
 - during labor, implications of 13
 - Protamine 50
 - Protective airway reflexes 109
 - Protein 152
 - binding 126, 219, 335
 - metabolism 264
 - S levels 138
 - Proteinuria 104
 - Prothrombin 265
 - Prothrombotic states 71
 - Proximal fracture femur 236
 - management of 236
 - Pseudocholinesterase production 276
 - Pseudomonas aeruginosa* infection 87
 - Public health 396
 - Pudendal nerve block 353
 - Pulmonary abnormalities 333
 - Pulmonary angiogram, computed tomography 523
 - Pulmonary arterial hypertension 270
 - Pulmonary artery 47, 57, 80
 - catheters 29
 - pressure 91
 - Pulmonary aspiration, risk of 156
 - Pulmonary atresia 45, 46
 - Pulmonary blood 48
 - flow 75
 - Pulmonary circulation 56
 - Pulmonary complications, postoperative 92
 - Pulmonary damage 196
 - Pulmonary diseases 129, 221
 - Pulmonary edema 84, 98, 138, 141, 199, 368, 479
 - cardiomegaly with 533f
 - re-expansion 103
 - Pulmonary embolism 4, 97, 98, 224, 522, 523
 - acute 523
 - risk factor for 255
 - suspected 523f
 - Pulmonary examinations 70
 - Pulmonary function tests 70, 87, 92, 254, 395, 396, 397f, 398
 - changes in 77t
 - limitations of 395
 - Pulmonary hemorrhage 402
 - Pulmonary hypertension 3, 4, 9, 142, 155, 333, 532f
 - reactive 3
 - severe 9
 - Pulmonary involvements, types of 252
 - Pulmonary microemboli 155
 - Pulmonary oligemia 49
 - Pulmonary stenosis 210
 - Pulmonary systolic pressures 9
 - Pulmonary torsion 84
 - Pulmonary vascular resistance 48, 52, 123
 - Pulmonary vasculature 216, 527
 - Pulmonary vasoconstriction 141
 - Pulse 196
 - algorithm 515f
 - generator 59
 - oximetry 49, 138, 201
 - pressure variation 278
 - volume recording 71
 - width, significance of 556
 - Pupil
 - reactivity score 166
 - unequal 172
 - Pupillary light reflex 226
- Q**
- Quincke-Babcock needle 551
 - Quinidine 11
 - toxicity 523
- R**
- Radial nerve 291
 - Radiation 66
 - RAE-Flex tubes 467
 - Ramp position 177f
 - Rashes 149
 - Raynaud's phenomenon 69
 - Rebreathing 452
 - circuit 453t

- depends 453
 - effects of 453
 - factor affecting 452
 - Red blood cells, transfusion of 147
 - Redivac systems 102
 - Reflux symptom index 179
 - Regional anesthesia 65, 73, 148*t*, 149, 202, 222, 261, 238, 283, 338
 - advantages of 328, 549
 - disadvantages of 328
 - instruments 549
 - role of 8, 40, 260, 356
 - techniques 220
 - Regional block 292
 - Regions of heart 504
 - Regulated cylinder pressure lower 415
 - Reinflate cuff 292
 - Remifentanyl 220, 261, 336
 - Renal and hepatic effects 219
 - Renal blood flow 343
 - Renal clearance 366
 - Renal dysfunction 73, 226, 315, 343
 - Renal effects 282
 - Renal failure 36, 199
 - acute 197
 - causes of 334
 - chronic 560
 - Renal function 40, 218, 275
 - impaired 227, 342
 - reduced fetal 139
 - test 87, 336
 - Renal insufficiency 294
 - Renal manifestations 42
 - Renal osteodystrophy 333
 - Renal perfusion pressure 343
 - Renal plasma flow 125
 - Renal problems 163
 - Renal system 217, 224
 - Renal transplant 332, 340
 - surgery 339
 - Renal vascularity 218
 - Renovascular hypertension 42
 - Rescue face mask ventilation 132
 - Rescue ventilation techniques 177
 - Reservoir bag 422
 - function of 451
 - Reservoir face mask 570, 571*f*, 571*t*
 - disadvantages of 571
 - Reservoir systems 575
 - Residual volume, significance of 399
 - Respirable gas inlet 576
 - Respiration
 - artificial 441
 - phases of 387*f*
 - spontaneous 458
 - type of 498
 - Respiratory acidosis 379
 - causes of 381
 - Respiratory alkalosis 379
 - causes of 381
 - Respiratory arrests 205
 - Respiratory assessment 76
 - Respiratory bronchioles, enlargement of 215
 - Respiratory changes 141, 297
 - Respiratory complications 281
 - perioperative 78
 - Respiratory depression 115, 300
 - Respiratory distress 311
 - Respiratory distress
 - causes 311
 - syndrome 53
 - acute 141, 193, 533
 - Respiratory drive 216
 - Respiratory failure 53
 - causes of 380
 - postoperative 368
 - types of 380, 380*t*
 - Respiratory infections 48, 72, 207
 - Respiratory insufficiency 84
 - Respiratory mechanics, monitor 392*f*
 - Respiratory optimization, preoperative 78
 - Respiratory paralysis 114
 - Respiratory rate 167
 - Respiratory responses 193
 - Respiratory system 93, 123, 156, 215, 216, 227, 275, 302, 315, 392, 595
 - changes 124*t*
 - compliance of 392
 - mechanics of 391
 - Respiratory tree 187
 - Resting echocardiography 23
 - Resting electrocardiogram 20
 - Restrictive lung disease 397
 - Restrictive pulmonary function tests, causes of 398
 - Resuscitation airways 491*f*
 - Resuscitators, types of 575
 - Retinal toxicity 247
 - Retrobulbar block 246
 - complications of 247
 - Retrobulbar needle position 246*f*
 - Retrosternal extension 301, 306
 - Reusable instruments 266
 - Reversible causes 478
 - Reye's syndrome, risk of 134
 - Reynolds number 421
 - Rhabdomyolysis 199, 205
 - Rheobase 554
 - Rheumatic fever, diagnose 5
 - Rheumatic heart disease 3
 - Rheumatoid arthritis 3, 178
 - Rhythm abnormalities 513
 - Ribs, absent 161
 - Right atrium 59*f*, 61
 - Right bundle branch block 510*f*, 514*f*
 - causes of 510
 - Right heart failure
 - signs of 48
 - symptoms of 48
 - Right ventricular failure 84
 - Right ventricular hypertrophy 5, 252, 509, 530*f*
 - causes for 519
 - Right ventricular outflow tract 46
 - Ring-Adair-Elwyn tube 211, 466, 467*f*
 - Ringer's lactate 157, 197, 198, 212, 297, 321, 348
 - solution 321
 - Ringer's solution 228
 - Risk, injury, failure, loss and end-stage renal disease (RIFLE) 344
 - Risser-Ferguson method 293*f*
 - Rivaroxaban 235
 - Robertshaw tube 469
 - Robot-assisted partial nephrectomy 285
 - Robotic cholecystectomy 280
 - Robotic surgeries, anesthetic considerations of 285
 - Robotic surgery 286
 - advantages of 285
 - device 285
 - types of 285
 - Robotic thoracic surgeries 286
 - Robotic-assisted surgery 342
 - Rocuronium 8, 260, 277, 335
 - Rollover test 105
 - Room air oxygen saturation 87
 - Ropivacaine 111, 352
 - Rotational thromboelastometry 143, 167
 - Rotterdam scale 170
 - Rubella infection 53
 - Ruben resuscitation valve 499
 - Ruben valve 499*f*
 - Runaway pacemaker 60
 - Rusch airway 493
- S**
- Sabrazes breath-holding time 93
 - Sacral canal contains 352
 - Saklad airway 493
 - Salicylates 365
 - Saline
 - epidural infusion of 112
 - normal 198, 516
 - Saphenous nerve 228
 - Sarcoidosis 179
 - Saturated vapor pressure 432
 - Schimmelbusch mask 438
 - Schonfeld's criteria 240, 240*t*
 - Schrader socket 416
 - Schwann cell distance 217
 - Sciatic nerve block 559*f*
 - Sclera and cornea 246
 - Scoliosis 161, 293
 - acquired 293
 - classify 293
 - congenital 293
 - idiopathic 293
 - severity of 293
 - surgery 300
 - Scope damage, causes of 180
 - Sebaceous glands 191
 - Secondary pressure regulators 416
 - Second-generation agents 317
 - Secretagogues 319
 - Sedation 148*t*, 171
 - Sedative 7
 - Sedative premedication 156
 - Seizures
 - prevent 113
 - recurrent 113
 - Selective serotonin reuptake inhibitors 553
 - Self-inflating bag 576
 - Sellick's maneuver 130
 - Semi-closed breathing systems 454
 - Sengstaken-Blakemore tube 270
 - Sensory nerve supply 246*t*
 - Septic shock 140
 - Serotonin 193, 204, 216
 - Serum
 - bilirubin 266
 - creatinine 320
 - measurement of 107
 - electrolytes 197, 201, 237, 320
 - enzymes 265
 - glutamic oxaloacetic transaminase 104
 - glutamic pyruvic transaminase 104
 - magnesium 114
 - sodium systemic effects 230*t*
 - trypsin 142
 - urea and creatinine 201
 - Setting sun sign 153
 - Sevoflurane 85, 335, 424
 - Shock 62
 - and circulatory collapse, causes of 129
 - energy 477
 - Shunt 49, 56, 154
 - and dead space 375
 - equation, classic 375
 - flow-regulating 154
 - insertion of 154, 164
 - left-to-right 52-54, 56
 - malformation
 - signs of 158
 - symptoms of 158

- malfunction
 - signs of 158
 - symptoms of 158
 - procedures, common complications of 155
 - surgery 49
 - types of 154, 155
 - ventriculoatrial 154
 - ventriculoperitoneal 154, 156
 - ventriculopleural 154
 - Shutoff valve 416f
 - Shuttle-walking test 77, 593
 - Shy-Drager syndrome 316
 - Sickle cell disease 289
 - Silhouette sign 531
 - Simple oxygen mask 570, 570f
 - advantages of 570
 - disadvantages of 570
 - Single breath count test 93
 - Single parameter tests 174
 - Single vocal cord paralysis 402
 - Single-bottle drainage system 100, 100f
 - Single-chamber demand pacing 59
 - Single-gene mutations 161
 - Sinus
 - arrhythmia 513
 - bradycardia 514, 514f
 - causes of 514, 515
 - node cells 214
 - node dysfunction 58
 - rhythm 11
 - sagittal 151
 - tachycardia 54, 514, 515f
 - Sinusoidal intrahepatic causes 269
 - Six-minute walk test 77
 - Skin
 - functions of 192
 - microcirculation 55
 - Sleep
 - apnea, history of 257
 - studies, indications for 253
 - Sleep-disordered breathing 224
 - Sleeping habits 251
 - Slow vital capacity test 399
 - Small airway obstruction 398
 - Small capacity systems 570
 - Smoke
 - components of 195
 - inhalation, mild 196
 - Smokers, chronic 75
 - Smoking 70, 136, 144
 - before noncardiac surgery 23
 - cessation of 78
 - effects of 93
 - Snider's match test 93
 - Soda lime 423, 424
 - active component in 423
 - canister 424, 425
 - granules 423
 - newer 424, 425t
 - traditional 425t
 - Sodium 332
 - bicarbonate 478
 - nitroprusside 43, 200
 - Soft systolic murmurs 10
 - Soft tissues 527
 - Softseal laryngeal mask 540
 - Sokolow-Lyon index 519
 - Soluble insulins 318
 - Somnolence 199
 - Sonomatic confirmation of tracheal intubation 466
 - Sorbitol 229
 - Space of Disse 264
 - Speech development 210
 - Spider angioma 273, 273f
 - Spikes, absence of 64f
 - Spina bifida occulta 161
 - Spinal analgesia, types of 549
 - Spinal and epidural needles 550f
 - Spinal anesthesia 115, 131, 137, 550
 - Spinal block, total 131
 - Spinal catheters 550, 551
 - Spinal cord 160
 - damage 298
 - tumors 298
 - Spinal hypotension 132
 - Spinal integrity 300
 - Spinal needles 550
 - types of 550
 - Spinal taps 154
 - Spiral arteries 107
 - Spirometry 294, 396
 - based on 397
 - contraindications for 396
 - indications for 396
 - interpretation 399, 400f
 - values 88f, 398
 - Spontaneous bacterial peritonitis 272
 - Spontaneous breathing 387, 455, 458f
 - and controlled ventilation 455, 458
 - Sputum 87
 - Square wave stimulus 560
 - Stable angina 20
 - Stair climbing 77
 - Standard resuscitator 576
 - Staphylococcus*
 - aureus* 86, 158, 211
 - epidermidis* 158
 - Static and dynamic lung volumes 397
 - Statins 27, 33
 - decrease 72
 - Steam sterilization 475
 - ST-elevation myocardial infarction 28, 520f
 - Stem bronchus, left main 469
 - Stenotic valvular disease, severe 10
 - Sterilization 585
 - Sterilize manual resuscitator 578
 - Sternal compression, depth of 477
 - Sternomental distance 175
 - Steroid 79, 94, 130, 341
 - treatment regimen, perioperative 342f
 - Stewart approach 378, 379t
 - Stickler syndrome 208
 - Stimulating facial nerves 562f
 - Stimulating needle 556
 - Stimulating ulnar nerves 562f
 - Stimulation, patterns of 563f
 - Stimulus frequency 555
 - Stool stercobilin 266
 - Storing compressed gas cylinders 400
 - Streamlined liner of pharynx airway 544f
 - Stroke 69, 73, 314
 - acute 327
 - Structural kidney injury 269
 - Subarachnoid block 110, 232
 - Subclavian artery 47
 - Subclinical hyperthyroidism 303
 - Subclinical hypothyroidism 303
 - Subcutaneous emphysema 85
 - Subcutaneous insulin 320
 - Subcutaneous nodules 5
 - Subcutaneous tissue 203
 - Subdural hematoma 172
 - Sublingual nifedipine 114
 - Submucosal cleft 208
 - Substance P 193
 - Substantia gelatinosa 204
 - Sub-Tenon's block 247
 - Succinylcholine 65, 138, 260, 335
 - action of 276
 - Sufentanil 115, 220, 261
 - Sugar control 321
 - Sulfonylureas 319
 - Sulfur dioxide 195
 - Sumatriptan 112
 - Superinfection 130
 - Superior laryngeal nerve, external branch of 312
 - Supplemental oxygen 165
 - Supportive treatment 142
 - Supraclavicular brachial plexus block 290
 - Supraglottic airway 189, 478
 - device 132, 178, 186, 534, 538
 - technique of 540
 - Supramaximal stimulation 560
 - Surgery
 - influence, type of 19
 - timing of 48
 - Surgical repair, complete 48
 - Surgical technique 295
 - Suxamethonium 202, 364
 - Sweat glands 191
 - Sydenham's chorea 5
 - Sympathetic block success 73
 - Sympathetic control, tests for 316
 - Sympathetic stimulation 109
 - Symptomatic bradycardia 58
 - Symptomatic pleural effusions 99
 - Synchronized intermittent mandatory ventilation 387
 - Syncope 113
 - Syndrome of inappropriate antidiuretic hormone secretion 169
 - Syndrome X 314
 - Systemic carbon anhydrase inhibitors 249
 - Systemic disease 178
 - pulmonary involvement in 401
 - Systemic embolism 4
 - Systemic hypertension 57
 - Systemic intoxication injuries 195
 - Systemic lupus erythematosus 3
 - Systemic to pulmonary shunts 47f
 - Systemic toxicity 113, 205
 - Systemic vascular resistance 47, 52, 123
 - Systolic murmur 45, 46
 - Systolic velocity 153
- T**
- Tachyarrhythmias 56, 516
 - Tachycardia 73, 128, 129, 138, 143, 148, 179, 222, 324
 - adult 516f
 - supraventricular 58, 224, 275
 - Tachyphylaxis 138
 - Tachypnea 48
 - Tacrolimus 341
 - Takayasu's arteritis 69
 - Tapentadol 340
 - Target organ damage, signs of 37
 - Target-controlled infusions, caution with 367
 - Temperature
 - compensation, techniques of 435, 440
 - control 157
 - monitoring 138, 201
 - probe 80
 - Temporary pacing 64
 - Temporomandibular joint 179
 - function, assessment of 174
 - Tensile test 409
 - Tension pneumothorax 196, 468, 485
 - Teratogen 53, 137
 - Teratogenicity 304
 - Terminal half-life, clinical application of 364

- Testing spinal integrity 297
 - Tetanic stimulation 562
 - fade on 563
 - Tetraiodothyronine 302
 - Tetralogy of Fallot 45, 46*f*
 - pathophysiology of 46
 - Theophyllines 112
 - Therapeutic catheterization 55
 - Thermoregulation 193, 217, 241
 - Thiamine deficiency 275
 - Thiazides 38
 - Thiazolidinediones 318
 - Thiopental 260
 - Thiopentone 79, 138, 257, 292, 321, 335
 - sodium 220
 - Third stage of labor, management of 148
 - Thoracic epidural analgesia 84
 - Thoracic-abdominal compression-decompression 485
 - Thoracoscopy 294
 - Thoracotomy 294
 - Thorax
 - changes 215
 - computed tomography 87
 - Three-bottle drainage system 101, 101*f*
 - Three-compartment model 363
 - Thromboangiitis obliterans 69, 69*t*
 - Thrombocytopenia 146
 - Thromboelastography 143
 - Thromboembolism 52, 240
 - Thrombophlebitis 155
 - Thromboprophylaxis 138, 237, 241
 - postoperative 241
 - Thumb, movement of 561
 - Thymectomy 286
 - Thyroglobulin 301
 - Thyroid 174, 183
 - function 305
 - tests 302, 303, 307
 - hormone 301, 303
 - actions of 302
 - status 301
 - malignancies 301
 - mass 307
 - large 301, 306
 - peroxidase 301
 - steal 305
 - stimulating hormone 303
 - storm 305
 - surgery 309, 311, 312
 - swelling 301
 - Thyroidectomy 304
 - with hyperthyroidism, anesthesia management for 304
 - Thyromental distance 174
 - Thyroxin 218, 302
 - Tibial nerve 561
 - posterior 552*f*
 - Tidal volume, variation in 567*f*
 - Tight sugar control 327
 - Timolol 249
 - Tissue asphyxia, causes of 200
 - Tobacco abuse 215
 - Tobramycin 249
 - Tocolytic therapy, role of 138
 - Tongue
 - displacement, mouth for 174
 - large 306
 - Tonsillar position 349
 - Tonsillectomy 213, 346, 347, 350
 - contraindications for 346
 - indications for 346
 - management of 349
 - surgery 347
 - Tonsillitis 346
 - history of 347
 - recurrent 346
 - Torkildsen shunt 154
 - Torsade de pointes 518*f*
 - Total body surface area 198
 - Total dose infusion 121
 - Total hip replacement 236
 - Total intravenous anesthesia 298, 366
 - Total knee replacement surgery 65
 - Toxic reactions 113
 - Toxicity, management of 113
 - Trachea 308
 - compression of 308
 - Tracheal intubation 132
 - contraindications of 464
 - Tracheal lumen 82, 471*f*
 - Tracheal mucosa 464
 - Tracheal stenosis 402
 - Tracheal tube 463
 - connector 494
 - Tracheomalacia 183, 402
 - management of 309
 - Tracheostomy
 - controlled 308
 - mask
 - advantages of 570
 - disadvantages of 570
 - surgical 166
 - tubes 489
 - Tramadol 116, 336
 - Tranexamic acid 231
 - Transarterial techniques 290
 - Transcatheter closure 55
 - Transcranial Doppler 153
 - Transcutaneous electrical nerve stimulation 66, 258
 - Transesophageal atrial pacing 64
 - Transesophageal pacing 64
 - Transfusion trigger 295
 - Transient ischemic attack 15, 253
 - Translaryngeal block 180
 - Transnasal humidified rapid-insufflation ventilatory exchange 188, 256
 - Transoral robotic surgeries 286
 - advantages of 286
 - Transthoracic echocardiogram 523
 - Transtacheal jet ventilation 186
 - Transurethral resection of prostate
 - management of 232
 - syndrome, treatment for 232
 - Transvenous pacemaker, indications for insertion of 513
 - Transvenous pacing 59
 - Trauma 145, 178
 - Traumatic brain injury 165, 171, 172, 327
 - Traumatic laryngeal fracture 465
 - Traumatic parenchymal lesions 172
 - Treacher Collins syndrome 208, 209, 209*f*
 - Treadmill 594
 - Trendelenburg position 283, 285
 - Tricarboxylic acid cycle 200
 - Tricuspid stenosis 5
 - Triexponential decline 363*f*
 - Trifascicular block 58, 513
 - Trigeminal nerve 212
 - Trilene 439
 - interlock 439
 - Tropicamide 259
 - Trueview laryngoscope 588, 589*f*
 - blade 589*t*
 - Tube advancement, method of 581
 - Tuberculosis 118
 - Tumescent local anesthesia 203
 - Tumor 145, 469, 529
 - embolization of 82
 - intratracheal extension of 307
 - necrosis factor 193
 - Turbulent flow 421
 - Two-bottle drainage system 101, 101*f*
 - Two-finger chest compression technique 485*f*
 - Tyrosine kinase inhibitors 305
- U**
- Ulnar nerve 291
 - Umbilical vein carries 52
 - Unipolar pacing 59, 60*f*
 - Universal circuit 459
 - Upper airway
 - edema 194, 394
 - exhalation 186
 - fixed obstruction of 402, 402*f*
 - obstruction 183, 195
 - Upper limb 203, 558
 - motor supply areas of 558*t*
 - Upper lip bite test 174
 - Upper lobe collapse, right 531*f*
 - Upper respiratory tract infection 48
 - chronic 208
 - Upward gaze palsy 153
 - Urea 229
 - Uric acid, elevated 105
 - Urinary albumin excretion 37
 - Urinary bladder 148
 - Urinary catheter 167, 337
 - Urinary incontinence 153
 - Urinary kallikrein excretion, decreased 106
 - Urinary tract, ultrasound of 162
 - Urine 71
 - albumin 104
 - analysis 104
 - anion gap 381
 - bile salts 266
 - bilirubin 266
 - output 138, 197
 - routine 162
 - urobilinogen 266
 - Urological surgeries, robotic-assisted 285
 - Uterine anomalies 145
 - Uterine arteries 106
 - Uterine atony 145
 - Uterine contractility 134
 - Uterine effects 114
 - Uterine hysteroscopy 66
 - Uterine inversion 146
 - Uterine müllerian anomaly 136
 - Uterine over distention 144
 - Uterine polyps 136
 - Uterine rupture 143, 144
 - Uterine serosa 148
 - Uterine tone 11, 141
 - Uterine trauma 145
 - Uterus 107, 131
- V**
- Vaginal delivery 12
 - Vaginal tear 143
 - Valsalva maneuver 316
 - Valve
 - anatomy and function 48
 - replacement 11
 - types of 9
 - unidirectional 422, 422*f*
 - Valvular heart disease 14, 214
 - Valvular surgery 11
 - van Lint method 246

- Vancomycin 12
- Vapor
 - and gas 432
 - compression cycle 416
 - pressure 432
 - produced, amount of 433
- Vaporization, method of 434
- Vaporizer 432, 449
 - arrangement of 449
 - capability 432
 - classify 433
 - concentration 432
 - efficiency 432
 - hazards of 437
 - in-system 436*t*
 - kind of 447
 - modern 449
 - newer 448
 - out-of-system 436*t*
 - output 432, 449
 - safety features of 437
 - use of 449
- Vascular causes 4
- Vascular endothelial growth factor 105
- Vascular functions 264
- Vascular occlusion 56
- Vascular stiffness 214
- Vasculitis 304
- Vasodilators 55, 115
- Vasopressors 146
- Vecuronium 8, 202, 260, 276, 277, 335
- Velocardiofacial syndrome 208
- Velopharyngeal insufficiency 210
- Vena cava
 - inferior 17*f*
 - superior 17*f*, 52
- Venous ophthalmodynamometry 153
- Venous return, decreased 281
- Venous stasis 227
- Venous thromboembolic disease 139
- Ventilation 57, 157, 168, 382
 - controlled 79, 456, 458
 - high-frequency 393
 - intraoperative spontaneous 158
 - intraoperatively 80
 - maintaining spontaneous 184
 - mode of 386, 393, 394
 - perfusion scan 142
 - phases of 387
 - postoperatively 83
 - pressure support 387
 - pressure-controlled 387, 388, 389*t*
 - prolongs 171
 - spontaneous 163, 184
 - volume-controlled 387, 388, 389*t*
 - working-controlled 502
- Ventilator 385
 - automatic 386
 - double circuit 385
 - high-powered 385
 - low-powered 385
 - single circuit 385
- Ventilatory management 196
- Ventilatory strategy 170
- Ventricular atrial shunts 155
- Ventricular dysrhythmias 171
- Ventricular fibrillation 480, 517, 518*f*
- Ventricular peritoneal shunts 155
- Ventricular septal defect 52
- Ventricular tachycardia 58, 275, 480, 516, 517
- Ventriculostomy, third 154
- Venturi effect 572
- Venturi mask 572, 573*t*
- Videolaryngoscope 581
 - advantages of 582, 583
 - limitations of 583
 - principle of 581
 - role of 584
- Videolaryngoscopy 581, 584
 - complications of 583
- Vili 381
- Viral infections, pediatric 134
- Visual backing disorder 153
- Visual confirmation 181
- Visual evoked potential 298
- Visual examination 174
- Visual inspection 174
- Visual loss, postoperative 299, 300
- Vital capacity, significance of 399
- Vitamin
 - B1 38
 - B12 119
 - D absorption 218
 - K 265
 - antagonists 235, 265
 - dependent factors 265
 - therapy 265
- Vocal cord paralysis, bilateral 402
- Volatile anesthetics 8
- Volatile inhalation agent 134
- Volutrauma 381
- Vomiting 149, 199, 283
- von Graefe's sign 302

W

- Wake-up test 298
- Warfarin 10, 14, 72, 242
- Warm air blankets 157
- Waters airway 490
- Wegener's granulomatosis 179
- Wenckebach phenomenon 512
- Wernicke's encephalopathy 275
- Wheeze test 92
- Whipple disease 3
- White blood cell 219
- White cell count 320
- White coat 37
 - hypertension 37
- White double-lumen tube 470*f*
- White tube 469
- Wide bore cannula cricothyrotomy 186
- Wilkins mitral valve score 8
- Williams airway intubator 491
- Wilsons scoring system 175
- Wolff-Parkinson-White syndrome 507
- Wood metal 411
- Working pressure 447
- Wound
 - care 241
 - hematoma 311
 - formation 13
 - infection, surgical 217
 - inspection, facilitate 197

X

- Xenon gas-filled bulbs 495

Y

- Yankauer's mask 438
- Yoke block, uses of 414
- Yoke blocks 414

Z

- Zieve's syndrome 274

Objective ANESTHESIA Review

A Comprehensive Textbook for the Examinees

Salient Features

- A comprehensive review book on anesthesia
- Provides a fully revised and updated text as per the latest evidence of the management of various cases as well as the new guidelines
- Presents the text in an easy-to-follow question-answer format
- Thoroughly discusses the equipment and table viva topics, such as arterial blood gases (ABGs), electrocardiograms (ECGs), pulmonary function tests (PFTs), cardiopulmonary exercise testing (CPET), etc. normally encountered in the examination
- Includes all the complicated cases and the entire range of anesthesia equipment
- Contains new chapters on Management of Acute Postoperative Pain, Cardiopulmonary Exercise Testing and Videolaryngoscopes
- Useful for the postgraduate examinees of anesthesia and also the practising anesthesiologists.

Atul Prabhakar Kulkarni MD (Anesthesiology) is currently Professor and Head in the Division of Critical Care, Department of Anesthesiology, Critical Care and Pain at the Tata Memorial Hospital, Homi Bhabha National Institute, Mumbai, Maharashtra, India. A recognized postgraduate teacher for MD (Anesthesiology), he is now a teacher for DM (Critical Care Medicine). A postgraduate diploma holder in Post Graduate Diploma in Health and Healthcare Management, he has received training in Intensive Care at St George Hospital, Sydney in 2002–2003. He has participated in many conferences on critical care and anesthesiology at international, national, and state levels. He was the Organizing Secretary for anesthesia review course since 2004 to 2018. He was Treasurer of Indian Society of Critical Care Medicine during 2004–2010. He was later General Secretary of Indian Society of Critical Care Medicine (2010–2014) before he took over as President of Indian Society of Critical Care Medicine (2015–2017) and the Chancellor Indian College of Critical Care Medicine. He was a Member of the Editorial Board of Indian Journal of Critical Care Medicine (IJCCM) and reviewer for IJCCM and Indian Journal of Anesthesia (IJA). He is also the past President of Association of South Asian Association for Regional Cooperation (SAARC) Critical Care Societies and Secretary General, and now he is the honorary Secretary General of the Asia Pacific Association of Critical Care Medicine. Currently he is the Editor-in-Chief of the Indian Journal of Critical Care Medicine.

Jigeeshu Vasistha Divatia MD (Anesthesiology) is Professor and Head, Department of Anesthesiology, Critical Care and Pain at the Tata Memorial Hospital, Homi Bhabha National Institute, Mumbai, Maharashtra, India. A postgraduate diploma holder in medicolegal systems, he has received training in Intensive Care at the St George Hospital, Sydney in 1995–1996. He is a past President of the Indian Society of Critical Care Medicine. He is also the past Editor-in-Chief, Indian Journal of Anesthesia (IJA). He was the Chairman, Scientific Committee of ISACON 2011, Mumbai. He was a Member Secretary of the Institutional Review Board of the Tata Memorial Hospital, Mumbai. He has been conferred with the Fellowship of the ISCCM (FISCCM), the Indian College of Critical Care Medicine (FICCM), and American College of Critical Care Medicine (FCCM). He has been member of the Expert Group for Standard Treatment Guidelines in Critical Care, Ministry of Health, Government of India. He has over 120 publications to his credit in national and international journals. He is Member of Editorial Board, Indian Journal of Anesthesia (IJA) and Indian Journal of Critical Care Medicine, and reviewer for national and international journals. He has presented many papers in several international, national, and state level conferences.

Vijaya Prakash Patil MD (Anesthesiology) Dip (Hosp Adminis) is Professor, Department of Anesthesiology, Critical Care and Pain at the Tata Memorial Hospital, Homi Bhabha National Institute, Mumbai, Maharashtra, India. She is a recognized postgraduate teacher, examiner, and papersetter for MD (Anesthesiology). She has delivered many lectures on critical care and anesthesia in several national and state level conferences. She is Organizing Secretary for Tutorials in Hemodynamic Monitoring and Therapy in Critical Care, an annual continuing medical education (CME) since 2004 to till date. She was West Zone Member of the Indian Society of Critical Care Medicine for the period of 2010–2014 and is currently the Secretary of the Indian Society of Critical Care Medicine. She is an Associate Editor of Indian Journal of Critical Care Medicine (IJCCM) and a reviewer of Indian Journal of Anesthesia (IJA).

Available at all medical bookstores
or buy online at www.jaypeebrothers.com



JAYPEE BROTHERS
Medical Publishers (P) Ltd.
www.jaypeebrothers.com

Join us on [facebook.com/JaypeeMedicalPublishers](https://www.facebook.com/JaypeeMedicalPublishers)

Shelving Recommendation
ANESTHESIA

ISBN 978-93-90020-48-5



9 789390 020485